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*of the*
# American Veterinary Medical Association

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H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.



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## INTERNATIONAL VETERINARY CONGRESS

"Going to Europe this summer?" is a question frequently heard when veterinarians get together nowadays. Judging from the letters received the past month, quite a number of members of the American Veterinary Medical Association are seriously thinking about a trip abroad this summer. Of course, the thing of particular interest this year is the Thirteenth International Veterinary Congress, which will be held at Zurich-Interlaken, Switzerland, August 20-27.

The dates for the Diamond Jubilee meeting of the American Veterinary Medical Association—July 5-6-7-8-9—were fixed with a view to accommodating those who will participate in the Official Tour of Europe sponsored by the A. V. M. A. The party will sail from New York, July 13, on the S. S. *Manhattan.* Dr. Adolph Eichhorn will be in charge of the professional program of the tour, and the business management and sight-seeing will be under the direction of Treasure Tours, Inc.

The party will return on the S. S. *Washington,* arriving in New York, September 15, after having visited London, Brussels, Scheveningen, Berlin, Leipzig, Dresden, Munich, Budapest, Vienna, Venice, Milan, Zurich, Interlaken, Geneva, Grenoble, Nice and Paris. The price of the tour is $794.00.

Another tour, slightly shorter in duration and costing only $614.00, has been arranged by Thos. Cook & Son, under the leadership of Dr. D. M. Campbell, of Chicago. Those who participate in this tour will depart from Montreal on July 23 and, after traveling 12,238 miles during a period of 55 days, will arrive in New York, September 8. Points to be visited include Glasgow, the Trossachs, London, Copenhagen, Berlin, Hanover, Amsterdam, Brussels, Paris, Zurich, Interlaken, Milan, Trieste, Naples, Algiers, Gibraltar, Lisbon and the Azores.

A third tour in connection with the Congress in Switzerland, also under the auspices of Thos. Cook & Son, will be under the personal direction of Dr. T. W. M. Cameron, of McGill University. The itinerary of this tour will include London, Zurich, Interlaken, Wiesbaden, Cologne, Brussels, Paris, the English lake region, the Trossachs, Edinburgh and Glasgow. This tour will consume 45 days, August 5 to September 18, and will cost $495. The party will depart from Montreal and will arrive back in the same city.

Dr. Adolph Eichhorn, chairman of the National Committee for the United States, is very desirous of obtaining a creditable number of memberships from the veterinary profession in this country. The membership fee has been fixed at 30 Swiss francs (approximately $7.00). The fee for lady members is 5 francs ($1.25). Each member will receive a copy of the complete Proceedings of the Congress, and those who attend will participate in all of the functions being arranged. Blank applications for membership may be obtained from the Secretary of the National Committee, Dr. H. Preston Hoskins, 221 N. La Salle Street, Chicago, Ill.

## INVITATIONS FOR 1939 CONVENTION

San Francisco has been on record for some time as wanting the 1939 convention of the American Veterinary Medical Association. However, due to the fact that the Golden Gate International Exposition will be held in San Francisco in 1939, there is considerable doubt of our ability to make satisfactory arrangements for hotel accommodations while the Exposition is in progress.

The invitation of San Francisco was very carefully considered by the Executive Board, at the recent meeting, but no action was taken, pending further investigations of the hotel problem. In the meantime, unusual interest has been shown in the 1939 convention and no less than four other cities in various parts of the country have entered the field. It has been suggested that the

invitation of San Francisco for 1939 be withdrawn and the 1940 meeting be held in that city, with the 1939 meeting held in a city more centrally located. Incidentally, at this point, we might mention the fact that Los Angeles wants the Fourteenth International Veterinary Congress, which is due to be held in 1942.

At the meeting of the Wisconsin Veterinary Medical Association, held in Madison, on January 12-14, it was decided to renew the invitation of the Wisconsin veterinarians for the American Veterinary Medical Association to meet in Madison in 1939. At the meeting of the State Veterinary Medical Association of Texas, held in Houston, on January 13-14, the veterinarians of the Lone Star State went on record as favoring Dallas for the 1939 A. V. M. A. convention. At the meeting of the Colorado Veterinary Medical Association, held in Denver, on January 20, it was decided to invite the A. V. M. A. to meet in that city in 1939. At the meeting of the Indiana Veterinary Medical Association, held in Indianapolis, on January 25-27, the Hoosier veterinarians decided that it was about time that the A. V. M. A. met in their state again, and decided to go after the 1939 meeting.

So, we have no less than five cities, in five widely separated states, in the field for the 1939 convention. There is even the possibility that other invitations are under consideration. The field is wide open.

## EXECUTIVE BOARD ELECTIONS

The special election held in Executive Board District 7 (Washington, Oregon, Idaho, Montana, Wyoming, North Dakota, South Dakota, Nebraska, Alaska, Hawaii and the Philippine Islands) came to a close on January 17. The winner was Dr. W. A. Sullivan, of Cheyenne, Wyoming, who will fill the unexpired term of Dr. C. H. Hays, namely, until the close of the 1940 convention. Dr. Sullivan received 46.6 per cent of the vote cast. Every member in Wyoming voted—something very unusual.

This is the year for regular Executive Board elections in District 2 (Pennsylvania, New Jersey and Delaware) and District 3 (Wisconsin, Illinois and Indiana). The early dates fixed for the 1938 convention created a rather unusual situation this year, with reference to Executive Board elections. With July 5 as the opening day of the meeting, it was necessary to start the election machinery on January 5—six months before the annual meeting. As only members in good standing are eligible to vote in A. V. M. A. elections, there were comparatively few so qualified on Jan-

uary 5, and the number of nominating ballots mailed to paid-up members was rather small. However, just as fast as members in the two districts have paid their dues, ballots have been sent to them. The polls for nominations will remain open until March 5.

## BEDTIME STORY TOLD AGAIN

Once upon a time, there was a veterinarian down in Texas who, on occasion, became more interested in other things (oil, for example) than veterinary medicine. Like scores of others, he did not keep posted on questions of vital interest confronting the veterinary profession. Other activities demanded his attention, for the time being, at least. Then, along comes the federal unemployment census. Things had not been going so well with our Texas colleague, so he filled in one of the census cards and mailed it to Washington. At the same time he wrote a letter to President Roosevelt, directing attention to the fact that county agricultural agents were engaging in veterinary activities and thereby contributing to unemployment among veterinarians. When the letter was received at the executive office in the White House, it was promptly referred to Dr. C. W. Warburton, Director of Extension Work. In turn, the latter gave the letter his official attention with equal promptness and the following communication (we almost said form letter) was forwarded to our friend in the Lone Star State

> Your letter of November 20 to the President has been referred to me for reply. If you will inform me about specific instances where county agricultural agents are performing veterinary services for farmers I assure you I will use my best efforts to correct the situation. We do not want extension agents to compete with veterinarians and frankly I think there are very few cases where this is happening. County agents everywhere are very busy on other duties, and most of them recognize they are not qualified to treat sick livestock.

Evidently not being at all familiar with the technic employed in such cases by those who are supposed to direct extension activities financed with federal tax money, our Texas veterinarian thought he saw an opportunity presented here for doing something really worth while. His next move was to ask a number of veterinarians to supply him with information based on instances of county agents invading the field of veterinary medicine.

Having had some little experience along this line over a period of fifteen years, we found it difficult to generate very much enthusiasm over the prospect of having an old wrong so easily made right. We figured that the chances were about a hundred

to one that a perfectly good idea would end in just about the same way that other similar ideas had—in nothing being done about it.

For the benefit of any others who may not be familiar with an old story, we will try to tell it briefly. Although agricultural extension activities are financed very largely with federal funds, and although these activities are supposed to be directed from Washington, this "remote control" is practically non-existent. Each year the state directors of extension proceed to the national capital for their annual conference with Director Warburton. Each of the 48 state directors tells Dr. Warburton what is going to be done in each state in the way of agricultural extension. These projects are formally approved, the conference is adjourned, and everybody goes home. And we are then informed, politely enough, that the individual states are left to manage such matters as the handling of animal diseases by county agents, on the theory that a state director of extension knows better what is needed in his own state than somebody seated in a swivel-chair, behind a desk, down in Washington, a thousand or more miles away. Sounds reasonable, does it not?

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## COMMITTEE ON LOCAL ARRANGEMENTS

President Brumley has appointed Dr. Cassius Way to act as chairman of the Committee on Local Arrangements for the Diamond Jubilee meeting of the American Veterinary Medical Association, to be held in New York City, July 5-6-7-8-9, 1938. Dr. Way has announced the complete personnel of the committee as follows

*Chairman:* Dr. Cassius Way, 25 Vanderbilt Ave., New York, N. Y.
*Vice-Chairman:* Dr. C. P. Zepp, 136 West 53rd St., New York, N. Y.
*Secretary:* Dr. Robert S. MacKellar, Jr., 329 West 12th St., New York, N. Y.

SUBCOMMITTEE CHAIRMEN

*Commercial Exhibits:* Dr. Robert S. MacKellar, Sr., 329 West 12th St., New York, N. Y.
*Educational Exhibits:* Dr. R. A. Hendershott, First Mechanics National Bank Bldg., Trenton, N. J.
*Small-Animal Clinic:* Dr. J. A. S. Millar, Box 318, Deal, N. J.
*Large-Animal Clinic:* Dr. A. G. Hall, 110 Hudson St., New York, N. Y.
*Entertainment:* Dr. Adolph Eichhorn, Pearl River, N. Y.
*Banquet, Reception and Dance:* Dr. Robert S. MacKellar, Jr., 329 West 12th St., New York, N. Y.
*Alumni Dinners:* Dr. J. E. Crawford, 708 Beach 19th St., Far Rockaway, N. Y.

# *Meet—*

LT. COL. R. A. KELSER

Of the Army Medical Research Board, Ancon, Canal Zone, who reports results of experiments on equine encephalomyelitis in this issue of the JOURNAL.

DR. CASSIUS WAY

New York City practitioner, who has been appointed chairman of the Committee on Local Arrangements for the A. V. M. A. Diamond Jubilee meeting in July.

DR. FRANK THORP, JR.

Of Colorado State College, who discusses oat hay poisoning in a paper published in this issue of the JOURNAL.

DR. CHAS. W. BOYER

Practitioner of Topeka, Kansas, author of paper on blood transfusion in dogs, appearing in this issue of the JOURNAL.

## APPLICATIONS FOR MEMBERSHIP

The number of applications filed during the past month fell just two short of the number filed during January, 1937. Of the 41 applications filed last month and given first listing below, we find nine credited to Minnesota, the results of the efforts of Dr. Joab P. Foster, A. V. M. A. Resident Secretary for Minnesota. Close behind the Gopher State we find California, with eight applicants, the results of the efforts of Dr. W. L. Curtis, A. V. M. A. Resident Secretary for California. The 24 other applicants are distributed throughout 14 states, the Canal Zone and Canada.

(See January, 1938, JOURNAL)

FIRST LISTING

ALLEN, FRED E. 1593 Hunter Ave., Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

ATTAWAY, RAY S. Box 168, Princeton, Ky.
D. V. M., Colorado State College, 1935
Vouchers: B. R. McCrory and I. E. Newsom.

BARCHFELD, WILLIAM P. 711 James St. N. S., Pittsburgh, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: L. D. Barrett and A. Henry Craige, Jr.

BAXTER, ERNEST C. 816 S. San Pedro St., Los Angeles, Calif.
D. V. M., Cornell University, 1930
Vouchers: W. L. Curtis and L. B. Wolcott.

BECKER, H. ROBERT 514 S. George St., York, Pa.
V. M. D., University of Pennsylvania, 1936
Vouchers: M. A. Emmerson and A. Henry Craige, Jr.

BURGESS, STOKES H. 605 N. 5th St., Mankato, Minn.
D. V. M., Chicago Veterinary College, 1915
Vouchers: Joab P. Foster and Wm. C. Prouse.

BURTON, JARED Wheaton, Minn.
V. S., Ontario Veterinary College, 1902
Vouchers: Joab P. Foster and William A. Billings.

CLAUSEN, LAWRENCE B. 1285 Sigel St., Saint Paul, Minn.
D. V. M., Chicago Veterinary College, 1914
Vouchers: Joab P. Foster and Vincent J. Robinson.

COLLINS, LT. HORACE R., JR. Fort Oglethorpe, Ga.
D. V. M., Kansas State College, 1937
Vouchers: Col. B. A. Seeley and Maj. S. G. Kielsmeier.

EARHART, ROBERT N. 42 Sheridan Ave., Fort Thomas, Ky.
D. V. M., Ohio State University, 1933
Vouchers: W. F. Guard and Leonard W. Goss.

FISHER, B. O. Great Falls, Mont.
D. V. M., Ohio State University, 1911
Vouchers: G. W. Cronen and H. F. Wilkins.

FISHER, VIRGIL H. Great Falls, Mont.
B. S., D. V. M., State College of Washington, 1937
Vouchers: G. W. Cronen and H. F. Wilkins.

FISHER, VURL E. 1323 E. Center St., Rochester, Minn.
D. V. M., Iowa State College, 1930
Vouchers: Joab P. Foster and Wm. C. Prouse.

GATHMANN, LT. RAYMOND A. Fort Oglethorpe, Ga.
D. V. M., Iowa State College, 1937
Vouchers: Col. B. A. Seeley and M. Jacobs.

HOGGAN, ROBERT W. 1285 Parkway Ave., Salt Lake City, Utah
D. V. M., Ohio State University, 1911
Vouchers: D. E. Madsen and E. D. Leiby.

JENNINGS, CHARLES G. Morris, Minn.
M. D. C., Chicago Veterinary College, 1901
Vouchers: Joab P. Foster and Wm. C. Prouse.

JONES, LELAND W. 156 E. 4th St. N., Provo, Utah
D. V. M., Colorado State College, 1932
Vouchers: E. A. Bundy and Oscar Wennergren.

KING, JOHN M. 222 N. 3rd St., Apt. 9, San Jose, Calif.
B. S., D. V. M., State College of Washington, 1930
Vouchers: W. L. Curtis and J. F. Horr.

MCFADDEN, GLENN M. Box 731, Daytona Beach, Fla.
D. V. M., Kansas State College, 1935
Vouchers: J. H. Yoder and Peter Garside.

MCGILVRAY, C. LEROY 183 Eglinton Ave. W., Toronto, Ont.
B. V. Sc., Ontario Veterinary College, 1935
Vouchers: J. A. Campbell and W. J. R. Fowler.

MCKAY, JOHN, SR. 2326 W. 2nd St., Duluth, Minn.
V. S., Ontario Veterinary College, 1895
Vouchers: Joab P. Foster and Henry C. Krueger.

MCLEAN, ROBERT T. 3684 Beverly Blvd., Los Angeles, Calif.
D. V. M., Kansas State College, 1933
Vouchers: W. L. Curtis and L. B. Wolcott.

MAURER, FRED D. University of Idaho, Moscow, Idaho
B. S., D. V. M., State College of Washington, 1937
Vouchers: E. E. Wegner and E. M. Gildow.

OPP, ALBERT L. Box 3032, Cristobal, Canal Zone
V. M. D., University of Pennsylvania, 1936
Vouchers: F. E. Lentz and J. Purdy Maugel.

PAUL, LT. EMMET W. 125 1st Ave., Salt Lake City, Utah
B. S., D. V. M., State College of Washington, 1937
Vouchers: Lt. Ernest L. Henkel and Lt. Col. John W. Miner.

PETERS, JOHN E. 11 Forest Ave., Arcadia, Calif.
D. V. M., Colorado State College, 1932
Vouchers: W. L. Curtis and J. W. Childs.

POLEY, LINN S. 2004 E. Quarrier St., Charleston, W. Va.
D. V. M., Colorado State College, 1937
Vouchers: P. M. Cellar and H. M. Newton.

QUORTRUP, ERLING R. Bear River Bird Refuge, Brigham, Utah
D. V. M., Texas A. & M. College, 1936
Vouchers: Don R. Coburn and W. H. Hendricks.

RAVEN, HOWARD C. 730 Hibbard Rd., Wilmette, Ill.
B. S., Connecticut State College, 1933
D. V. M., Iowa State College, 1937
Vouchers: O. Norling-Christensen and G. S. Elwood.

RIEDE, PETER H. Mabel, Minn.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: Joab P. Foster and Wm. C. Prouse.

ROBERTS, IRWIN H. Box 55, Yadkinville, N. C.
D. V. M., Cornell University, 1937
Vouchers: H. D. Pritchett and A. A. Husman.

ROCKEY, WILLIAM H. 430 College St., Woodland, Calif.
D. V. M., Kansas State College, 1935
Vouchers: W. L. Curtis and C. B. Griffiths.

ROOT, ROBT. R. Livermore, Calif.
D. V. M., San Francisco Veterinary College, 1915
Vouchers: W. L. Curtis and C. B. Outhier.

SATUREN, ISRAEL M. 309 N. Marshall St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr., and John D. Beck.

STOCK, LESLIE E. Kensington, Minn.
D. V. M., Iowa State College, 1929
Vouchers: Joab P. Foster and Henry E. Schwermann.

TAYLOR, C. EDWARD 230 State St., San Mateo, Calif.
B. S., D. V. M., State College of Washington, 1934
Vouchers: W. L. Curtis and C. B. Griffiths.

UPTON, JOHN R. 660 Fairview Ave., Arcadia, Calif.
D. V. M., San Francisco Veterinary College, 1915
Vouchers: W. L. Curtis and G. W. Foelschow.

WEBER, CLAUD M. Oakville, Ind.
D. V. M., Indiana Veterinary College, 1916
Vouchers: F. C. Tucker and J. L. Axby.

WESTFALL, JOHN H. Bristol, N. H.
B. V. Sc., Ontario Veterinary College, 1913
Vouchers: C. L. Martin and C. A. Bottorff.

WILSON, WARREN S. Buffalo, Minn.
D. V. M., Terre Haute Veterinary College, 1914
Vouchers: Joab P. Foster and Wm. C. Prouse.

YOUNG, CLARENCE C. Box 131, Nacogdoches, Texas
D. V. M., Texas A. & M. College, 1931
Vouchers: Chas. W. Neal and Matthew E. Gleason.

## Applications Pending

SECOND LISTING

(See January, 1938, JOURNAL)

Bruner, Dorsey W., 371 Linden Walk, Lexington, Ky.
Butler, Lt. Lucius N., Jr., CCC Army-1, Barksdale Field, La.
Errington, Burnard J., 311 S. Hanover Ave., Lexington, Ky.
Fechner, Lt. Walter W., Hdqrs. Dist. 'E" CCC, Camp Beauregard, La.
Kral, James, 19½ E. Crescent St., Augusta, Me.
Landsberg, Max E., c/o Kingfisher Hotel, Kingfisher, Okla.
Rasmussen, Frederick R., Glencoe, Minn.
Teick, Earnest R., Velva, N. Dak.
Wainwright, Charles R., Purcell, Okla.
Yow, Arch C., Henderson, N. C.

The amount which should accompany an application filed this month is $9.58, which covers membership fee and dues to January 1, 1939, including subscription to the JOURNAL.

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The first x-ray picture taken in America was credited to Professor E. B. Frost, of Dartmouth College, January 20, 1896.

## COMING VETERINARY MEETINGS

Virginia State Veterinary Medical Association and Conference for Graduate Veterinarians at Virginia Polytechnic Institute. Blacksburg, Va. February 1-3, 1938. Dr. A. J. Sipos, Secretary, 1102 State Office Bldg., Richmond, Va.

Connecticut Veterinary Medical Association. Hotel Bond, Hartford, Conn. February 2, 1938. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

Hudson Valley Veterinary Medical Society. Poughkeepsie, N. Y. February 2, 1938. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. February 16, 1938. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Illinois State Veterinary Medical Association. Hotel Abraham Lincoln, Springfield, Ill. February 17-18, 1938. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

Kansas City Veterinary Association. Auditorium, General Hospital No. 1 Annex, Kansas City, Mo. February 21, 1938. Dr. C. W. Schulz, Secretary, 800 Woodswether Rd., Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Building, Balboa Park, San Diego, Calif. February 21, 1938. Dr. E. F. Sheffield, Secretary, 3895 Pacific Blvd., San Diego, Calif.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 21-25, 1938. Dr. I. S. McAdory, Secretary, Alabama Polytechnic Institute, Auburn, Ala.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. February 23, 1938. Dr. C. S. Rockwell, Secretary, 4927 Osage Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. February 23, 1938. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Colorado State College Veterinary Short Course. Fort Collins, Colo. February 23-25, 1938. Dr. I. E. Newsom, Dean, Colorado State College, Fort Collins, Colo.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. March 1, 1938. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. March 2, 1938. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Saint Louis District Veterinary Medical Aessociation. Melbourne Hotel, Saint Louis, Mo. March 2, 1938. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Louisiana Veterinary Medical Association and Louisiana State University Veterinary Short Course. Dalrymple Memorial Building, Louisiana State University, Baton Rouge, La. March 2-3, 1938. Dr. C. M. Heflin, Secretary, Box 1933, Baton Rouge, La.

Houston Veterinary Association. Houston, Texas. March 3, 1938. Dr. Claude Canion, Secretary, 409 Link Rd., Houston, Texas.

Arkansas Veterinary Medical Association. Eastman Hotel, Hot Springs, Ark. March 7-8, 1938. Dr. T. M. Dick, Secretary, City Health Dept., Little Rock, Ark.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. March 8, 1938. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Willamette Valley Veterinary Medical Association. Portland, Ore. March 8, 1938. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. March 9, 1938. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Northwestern Ohio Veterinary Medical Association. Hotel Waldorf, Toledo, Ohio. March 10, 1938. Dr. C. W. Witty, Secretary, Elmore, Ohio.

Ak-Sar-Ben Veterinary Medical Association. Fontenelle Hotel, Omaha, Neb. March 14, 1938. Dr. J. D. Ray, Secretary, 1124 Harney St., Omaha, Neb.

Maine Veterinary Medical Association. University of Maine, Orono, Me. March 31, 1938. Dr. A. E. Coombs, Secretary, 1 Kennebec St., Skowhegan, Me.

American Veterinary Medical Association. Hotel Pennsylvania, New York, N. Y. July 5-9, 1938. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

# BLOOD TRANSFUSION IN DOGS*

*By* CHAS. W. BOWER, *Topeka, Kan.*

It has been wisely said that the blood is the "life-giving substance" of the body. When one stops to consider the composition and the purpose of the blood, then the foregoing statement can be taken almost literally.

Definitely speaking, transfusion of blood simply means a transfer of "living substance" from one person or animal to another. The writer, however, wishes to broaden this interpretation somewhat by including the transfer of blood from one part of the patient's body to another region of that same body, thus producing an auto-stimulation.

There are many different types of blood transfusion which will merely be mentioned at this time. For example: the direct vein-to-vein method, where the blood is drawn from the donor's vein and directly transferred into the vein of the recipient. In this instance, the donor's vein and that of the recipient are connected with an instrument consisting of a double-acting syringe or a by-valve and ordinary syringe, this, of course, being united to a needle in the donor's vein with rubber tubing and likewise into the vein of the recipient. If the quantity of blood is small, an indirect transfer may be made by using a series of sterile syringes. For convenience and saving of time, there should be two operators, one to draw the blood from the donor and a second operator to inject it into the recipient.

Another indirect method which has become very popular is the sodium citrate method of transfusion. Briefly, the technic of this method is to have a sterile container into which has been placed a given quantity of a 2½ per cent solution of sodium citrate. The vein is punctured and the blood is allowed to flow into the container of sodium citrate. An assistant should continuously agitate the vessel during the process of withdrawing the blood. When sufficient blood has been collected, it is injected into the patient either with syringes, with a pressure intravenous outfit, or by the by-valve and syringe method. Sodium citrate transfusion is of course subject to all the accidents and drawbacks incident to transfusion by any other method, and in addition there is the possibility that the blood may clot despite all precautions. This may be due to the fact that the blood and sodium citrate were not mixed with sufficient thoroughness or because there is not a high enough percentage of sodium citrate to mix with the blood.

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

Then again, there is a slight danger of getting a sodium citrate reaction in the patient. This may be noticed at first by merely chilling, but if it increases the patient will go into a tetany. This, undoubtedly, is a result of a chemical reaction between the chemicals of the natural blood and the sodium citrate. It is always wise to discontinue giving the citrated blood when a severe chill attacks the patient. It has been said that the percentage of reactions from sodium citrate transfusions is undoubtedly higher and probably more severe than with other methods of transfusion. Generally, however, they are not harmful in any way. Therefore, the possibility of their occurrence should not deter a competent operator from selecting this method, especially in view of the numerous advantages offered by this technic.

## Methods Compared

Probably the greatest disadvantage of the citrated blood method is one that might be credited as its greatest advantage, namely: the fact that it can be given slowly and the operator who is only slightly adept at transfusions will be able to use it. The fact that it is often given so slowly and the blood allowed to remain outside of the body unnecessarily long is apt to cause this method to fall into disrepute rather than be one of our greatest helps in medical and surgical therapy.

The chief disadvantage of the vein-to-vein method of blood transfusion is that the clotting period of the dog is so short and the blood flows so slowly from the vein, thus preventing free passage of blood through the transfusion outfit.

The artery-to-vein method has been recommended and used by some operators but here again the greatest difficulty is the rapid clotting of the blood which may take place in the canula, rubber tubing, or even within the blood-vessels themselves.

The most practical method of transfusing blood is from the heart of the donor to the vein, peritoneum, or even the heart of the recipient.

Contrary to the general belief of many, the heart method is much the simplest and the most humane to the donor. The donor is anesthetized under morphine or pentobarbital sodium, the latter being preferred. The heart is pierced with a 12-gauge needle which has been previously attached to a rubber tube and by-valve. The large needle does less harm to the heart muscle than a smaller one and once the blood flow has been established it will seldom stop from clotting. The patient or recipient should be placed near the donor, and may or may not be anesthetized, although I prefer some narcosis if the condition will permit. If the vein

route is used, a rather large needle is preferable, for example a 14- or 16-gauge, which tends to lessen the danger from clotting. The transfer of blood should not be made too rapidly and the amount should be governed by the suspected blood-pressure of the patient. Ordinarily 50 to 100 cc is sufficient by this route unless the recipient has lost large quantities of blood.

The greatest disadvantage of this method is the danger of emboli due to air or clot and also hemolysis due to the incompatibility of the two bloods.

In view of these major disadvantages, the writer has adopted the heart-to-peritoneum method in most cases and the heart-to-heart method in extreme cases. This latter method will be described more completely later. By the intraperitoneal method there is no danger of shock. Furthermore, owing to the slow absorption, it is not necessary to be so careful about the typing of blood.

Then again, it gives the advantage of increasing the leukocytes or producing a leukocytosis. Just why a leukocytosis does occur when whole blood is injected into the peritoneal cavity has never been definitely explained although some attribute its action to a non-specific protein formed from the blood-cells.

An additional advantage of the intraperitoneal route is the increase in dosage. Fifty to 75 cc can easily be injected into the peritoneal cavity of a half-grown kitten, or 150 to 200 cc may be given to a Boston terrier without danger. There may be some slight pain or distress for a few moments after the operation, but it is only transitory.

The heart-to-heart method is employed in cases of severe internal hemorrhage and shock where the blood-pressure is so low that the vein can not be raised. In such cases it is very desirable to inject the blood from the donor directly into the right heart of the recipient, thereby restoring the arterial pressure and in turn reëstablishing the venous volume.

## Indications

There are many indications for blood-transfusions in small animals. The most predominant is shock, particularly shock following internal hemorrhage. If blood is transfused into an animal that has lowered blood-pressure due to a hemorrhage, the pressure can be returned close to normal if the amount of blood injected is equal to the amount lost. Under these conditions the pressure remains near the normal point for a long time. If, how-

ever, physiological saline solution is used instead of whole blood, the rise of pressure is neither so great nor so sustaining.

The explanation as to why saline injections do not cause sustained rises of blood-pressure must be thought of in terms of osmotic pressure of the blood colloid. In most situations in the body the capillary walls are impermeable to the blood colloid, hence their low but constant osmotic pressure counteracts their capillary pressure and prevents the passage of liquid into the tissue. Physiological saline solution, however, contains no collodial material. Therefore, when it is introduced into the circulation it lowers the osmotic pressure of the blood colloid and a corresponding volume of liquid is soon pumped into the tissues and the rise of blood-pressure caused by its injection disappears. If acacia is added to the physiological saline solution to the extent of 6 to 7 per cent, the resulting solution known as gum saline causes a sustained rise in blood-pressure when injected. However, the intravenous use of acacia is not without danger.

Anemia is another indication for transfusion of blood. If the hemoglobin percentage is less than 85 per cent, blood-transfusion in some form should be offered. The writer prefers to use the heart-to-peritoneal-cavity method of transfusion on cases which are anemic from infectious diseases or slow convalescents.

In treating acute gastritis or enteritis of cats, intraperitoneal injections of 50 to 75 cc of blood from a healthy tom cat are very beneficial. It is not a panacea for this disease, but one is greatly encouraged by the sudden improvement of the patient from such therapy. Such infectious diseases as canine distemper are also materially helped by the injection of large quantities of healthy blood into the peritoneal cavity. Animals which are convalescing slowly can be greatly stimulated by peritoneal injections of whole blood. Furthermore, there are certain toxemias which respond to hemotherapy. Here again, the writer recommends intraperitoneal injections of transfused blood.

## Selecting the Donor

Just a word about the selection of the donor. In selecting the donor, the veterinarian should be careful to obtain a healthy animal, particularly one which is not a sufferer from any infectious disease. Care should be taken to examine the animal for *Demodex folliculorum* and also for *Dirofilaria immitis*, as it might be possible to transfer either of these parasites to the recipient. It has been said that the bloods of mongrels are compatible and also that the blood of a mongrel given to a pure-bred would be compatible, but that bloods of the same breed are often incom-

patible. If this be the case, it is safe to take blood from a mongrel for any breed and *vice versa*.

The most important factor to be determined is whether the serum of the recipient will agglutinate the blood-cells of the donor. This may be crudely determined by mixing a small amount of the recipient's serum with a few drops of a saline suspension of cells from the donor. If the blood of the recipient is incompatible with that of the donor, there will be clumping of the cells very similar to a positive reaction of the agglutination test for Bang's disease when the rapid plate method is used. When the intraperitoneal method is used, the question of incompatibility of the blood is no problem. However, if the vein-to-vein method is used, every precaution should be taken against using blood which is incompatible.

## Autohemotherapy

Before concluding a discussion on transfusion of blood in dogs, it appears fitting that we should at least mention autohemotherapy. It may or may not have a scientific basis, yet the results of this form of therapy have been encouraging in certain conditions.

The procedure consists of withdrawing, under sterile technic, from 1 to 5 cc of blood from the patient's vein and injecting it into the thigh muscles. This method of therapy has been used with great success in certain types of cutaneous infections, chronic eczemas, flat and papillomatous warts. It likewise has a value in combating certain infectious diseases. It has been proven that the blood-corpuscles have increased following the injection. This increase is particularly significant when we recognize that the blood-corpuscles aid in the formation of antibodies.

As to its manner of action, there is little agreement. The idea first advanced was that it was a form of protein shock therapy. But this conception is too simple to explain the nature of the effect of the injection of so complex a product. It has been shown that the blood is absorbed with great rapidity in about an hour, that is to say, much sooner than with foreign protein. The writer has used this on a number of cases and feels that he has obtained encouraging results. As an example, the following is offered as a case report:

## Case Report

*Patient:* Fox terrier, three years old, weight 25 lbs.

*History:* Had been fed a normal diet, consisting of meat, cereal and vegetables. It was not fed too heavily on vegetables or

cereals. During the summer months, he had been accustomed to lying among the evergreen shrubbery. For two weeks previous to our examination, small welts appeared over his body very much like the welts caused from the sting of a bee. At first, these welts were not sensitive; soon they began to itch and the itching became so intense that the skin was made raw all over the inner surface of the thighs.

*Treatment:* The animal was admitted to the hospital and examined thoroughly for fleas but none were found. A diet was planned so that it would be about the same as he had been getting at home. On the first day of the treatment, 2 cc of blood was drawn into a sterile syringe from the external saphena vein. This was immediately injected into the deep muscles of the thigh.

*Results:* Twelve hours following this injection, some of the welts appeared to diminish in size and the sores on the inner surface of the thighs began to dry and take on scab formation. Twenty-four hours after the injection, a most noticeable disappearance of the welts began. Three days after the first injection, a second injection was made after withdrawing 3 cc of blood. Twenty-four hours after this second injection, all of the welts had completely disappeared and the sores on the inner surface of the thighs had completely healed. Forty-eight hours after the second injection, the patient was discharged. If you will kindly note, in the treatment of this patient we refrained from giving any internal medication; also there was no external medicinal treatment. There was not even a bath given. We tried to keep the diet the same as he had been getting at home.

We have used this method of therapy on various animals that have been suffering from skin affections and have had exceedingly encouraging results. Remember please that this is not a panacea for skin diseases, but it is a therapy that is worthy of consideration and deserves more tests. I am hoping that others may find it convenient to use it and report their findings.

## SUMMARY

To summarize this discussion briefly, we find that there are two methods of transfusion of blood in dogs, namely, the direct and the indirect. Under the direct we have the choice of the vein-to-vein transfusion, heart-to-vein, or heart-to-peritoneal-cavity, the latter being preferable. In shock cases, particularly due to hemorrhage, transfusion from the heart of the donor directly into the heart of the recipient is recommended.

Under the heading of indirect transfusion we have chiefly the sodium citrate and blood method and also the transferring of blood from the donor to the recipient by a series of sterile syringes. The former is preferable. However, it might be accompanied by shock due to a chemical reaction of the sodium citrate and the blood.

The equipment used for transfusions consists of the pressure intravenous outfit, the series of sterile syringes, and the by-valve and syringe outfit, the latter being recommended by the writer.

Indications for transfusions in dogs are shock, internal hemorrhage, anemia, infectious enteritis, distemper, slow convalescing patients, and certain toxemias. Certain precautions should be taken in the selection of the donor. Only healthy, vigorous animals free from infectious diseases, *Demodex folliculorum* and *Dirofilaria immitis* should be chosen. There is no danger of incompatibility of the bloods when the intraperitoneal method of transfusion is used.

Autohemotherapy has been tried in animals, particularly those suffering from various types of skin ailments. Results have been very encouraging.

### Discussion

Dr. J. V. Lacroix: During the past decade, Lockhart and others in this country have stimulated an interest in the use of blood-transfusions in canine practice, but new methods are not soon adopted generally even when they are valuable. There are many indications for the use of blood-transfusions in canine practice and some indications for it in the treatment of cats.

Doctor Bower has nicely summarized the advantages and disadvantages of direct transfusion. In recent years, in medical practice, there has been a shift toward the indirect method of transfusion. The work of Yudin with cadaver-blood has tended to advance this field, in that stored blood was employed. To illustrate the extent to which the indirect method has been employed recently, I quote data from the Mayo Clinic (*Staff Proceedings*, vol. 10, p. 270). In 1934, 1,021 blood transfusions, probably all by the citrate method, were given; in 1935 and 1936, the same method was employed and in 1936, 2,058 transfusions were given to 1,073 recipients. Refrigerated blood was used in 149 cases. At the Cook County Hospital, Chicago, there has recently been established a blood-bank, where citrated blood is deposited and this blood is preserved by refrigeration.

While careless handling might cause the use of refrigerated blood to fall into disrepute, with careful handling and proper storage, this method offers many possibilities. It should mean that an ample supply of blood is available at all times and should make the process of transfusion more simple. The blood should be held at a temperature of 4 to 6° C. (approximately 40° F.). Yudin states that the blood is usable for a period of one month. The Mayo Clinic suggests twelve days.

Shock was reported by Yudin in 20 per cent of transfusions with citrated blood, as against 5 per cent with direct transfusion. Of the 2,058 transfusions at the Mayo Clinic (1936), chill and rise in tempera-

ture were noted in 16.2 per cent and bizarre reactions in 11.8 per cent. In the 149 transfusions with refrigerated blood, there were no untoward reactions. In 87.2 per cent, there were no reactions and only noticeable reactions in 4.8 per cent.

Melnick and Cowgill (*Proceedings of the Society for Experimental Biology and Medicine* (1937), vol. 36, pp. 697-700) state: "Although dogs are generally compatible with each other as regards blood-transfusions and this is evidenced by successful initial injections of cells, subsequent transfusions may create a sensitization which renders the donors incompatible thereafter. The development of isoagglutinins and isohemolysins results in the production of an intense hemoglobinuria; the continuation of the cell infusions, spaced from 7 to 10 days apart, is followed by symptoms of profound shock." In this study the authors reason that if it is true, as they have concluded, that 50 per cent of dogs belong to either the Alpha or Beta group and if these are the only two groups present in that species, it follows that there is only one chance out of four that an incompatibility will occur when a dog is subjected to repeated infusions of red blood-cells from a donor animal.

Probably this accounts for the absence of shock noted in laboratories where dogs are hyperimmunized. There cold blood is employed and incompatible blood is always diluted with blood that is compatible. Shock accompanying the administration of blood to dogs may result from an excess of sodium citrate. Anti-coagulants such as sodium citrate or sodium or potassium oxalate prevent clotting of the blood by removing the soluble calcium salts in the blood. Sodium citrate removes the calcium in the form of a soluble, unionizable calcium citrate. Sodium oxalate removes the calcium by precipitation and for this reason is contraindicated in transfusions. If there is an excess of anticoagulant in the blood for transfusion, a union will take place between the anticoagulant and the calcium in the blood of the recipient. For the prevention of coagulation, potassium or sodium oxalate is added in a concentration of 0.1 per cent after mixing. Sodium citrate is added so that the final dilution is 0.3 to 0.4 per cent. At this dilution, in our work, it has been impossible to prevent coagulation when blood is stored.*

Too often a 2½ per cent solution of sodium citrate is placed in a receptacle to which an unknown quantity of blood is to be added and there will be an excess of sodium citrate with tetany as a possible result, when this blood is administered. I do not profess to be able to distinguish between tetany of this sort and so-called shock resulting from other factors. However, I think it is a point worthy of mention.

I have had no experience in the transfusion of blood directly from the heart of the donor to the recipient. I hope that others who have will express themselves on the subject.

Doctor Bower is to be complimented for recommending the routine use of sedatives in this work. We have found no contraindications for use of barbiturates whenever we wish to tap the heart of a donor and since humanitarian aspects are to be considered in routine hospital practice, anesthesia is to be recommended. Also, narcosis facilitates the execution of a good technic in harvesting the blood.

At this point I will quote Mr. Howard J. Bly, who has had charge of this work in our hospital for about two years.

"Two one-liter flasks are hooked up in series to a water-flow suction pump to collect the blood. A short rubber tubing (about 10 inches) connects a 14-gauge bleeding-needle with the collecting-flask that contains the citrated solution. The second flask is an overflow to prevent any back-flow of water from the pump as may occasionally occur and thus contaminate the blood. The apparatus is, of course, sterilized in

*2% solution—1 part to 4 parts blood. (MacLeod, Physiological Chemistry.) 3% solution—1 part to 9 parts blood. (Amadon, *Univ. of Penn. Bulletin*, Vol. 37, No. 63, 1936.)

an autoclave and allowed to cool before being used. The donor is not fed for several hours before being bled.* It is anesthetized with pentobarbital sodium solution, given intravenously to the point of loss of reflex to pinching a toe. The area over the heart is clipped and cleansed. The animal is suspended in an upright position with the head slightly lowered. The area to be invaded is swabbed with tincture of iodine and the needle inserted into the chamber of the heart by pushing it through an intercostal space posterior to the olecranon in an upward and backward direction. If the animal is suspended in a proper manner, the blood will flow downward through the needle and tubing into the collecting flask so that the suction pump is necessary only to hasten the flow or to prevent formation of clots in the needle or tubing. As the blood flows into the collecting flask, this is gently rotated to mix the citrated solution thoroughly with the blood. Later it is tested for the presence of microfilaria or bacteria and bottled in 125-cc, rubber-stoppered serum bottles if negative. If, due to trouble in bleeding, danger of contamination is suspected, phenol in normal saline, q. s. 0.5 per cent, may be added. However, with its use more shock may be encountered and more hemolysis of the cells may result on standing.

In connection with the therapeutic use of blood, Bly reports that, "where trouble is encountered in administering blood intravenously, due to extreme nervousness on the part of the recipient, structural anatomic difficulties (short, crooked legs of Dachshunds and other breeds), low venous pressure and occasionally obliteration of the veins due to repeated daily intravenous injections, blood may well be given into the peritoneal cavity. The first cases we chose for this mode of administering blood were animals long sick from so-called distemper which were classed as moribund. Prior to this, animals that were held for euthanasia were given injections of blood intraperitoneally before euthanasia to check on the absorption of blood. In all cases it was found that absorption was satisfactory. Many of the critically sick animals treated showed the same favorable response that had been noted with intravenous medications. Thereafter, in addition, to the occasional anemic patient, animals suffering from so-called distemper complications, where there was vomiting and diarrhea, were chosen as being suitable for receiving blood intraperitoneally. Dosage was as much as 1 cubic centimeter per pound of body weight, daily."

There is need for greater care in intraperitoneal injections of blood than where blood is given intravenously. In the latter instance infection seldom results, whereas the peritoneal cavity, on the contrary, is quite vulnerable.

Doctor Bower has mentioned a practical test for determining incompatibilities of blood. Where some method is not used to determine compatibility, blood from two or more donors should be employed to lessen the likelihood of shock.

I am glad to note that Doctor Bower has interested himself in autohemotherapy. This therapy was advocated by Panisset, at Alfort, France, about ten years ago, but I have had too little experience in its use to arrive at any conclusions.

*In a personal communication from Dr. Andrew C. Ivy, he states, regarding work that was in progress in March, 1937: "This shows that to avoid all reactions to transfusions one not only has to cross-agglutinate, but also to fast both the donor and recipient or to feed both. In fact it is best to feed both, because we have never seen a reaction in dogs under such conditions, provided of course their bloods are compatible by the cross-agglutination test."

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Society waits unformed and is between things ended and things begun.—*Whitman.*

# BARBITURATES AS SAFE AND EFFICIENT GENERAL ANESTHETICS IN CANINE SURGERY*

*By* E. E. SWEEBE, *North Chicago, Ill.*
*Veterinary Department, Abbott Laboratories*

Hewitt, in his book entitled, "Anesthetics and Their Administration," published as far back as 1907, listed the following properties which an ideal anesthetic should possess:

1. It must be able to produce universal insensibility.
2. It must be capable of being readily introduced into the circulation without discomfort to the patient.
3. It must produce its effect gradually and progressively so that its action must be under control.
4. It must bring about not only general sensory paralysis but such a loss of motor power as to render practicable the performance of most delicate surgical operations.
5. Its action on the sensory and motor systems must not be associated with greater excitement, convulsive movement or interference with respiration, circulation or other important vital processes than can be controlled or corrected by the anesthetist.
6. Lastly, it is essential that the agent employed shall be of such a kind that when it has been withdrawn the whole organism shall resume its functions and return to that condition in which it existed immediately before the anesthetic was applied.

At this early date, practically only volatile anesthetics met these demands. Since this period, considerable progress has been made in chemistry and pharmacology which has resulted in the discovery and preparation of new compounds capable of producing anesthesia with the same degree of safety and efficiency in properly controlled dosage with more convenience to both patient and anesthetist.

The one main advantage of a volatile anesthetic is that a short time after its administration is discontinued, the anesthesia subsides, while the older forms of non-volatile anesthetics (such as chloral hydrate), if once administered, stayed in the body for a long period, so that introduction of anesthesia always meant a prolonged state of anesthesia and depression.

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

During the past ten years, research has resulted in changes in the chemical structure of non-volatile anesthetics and the development of new compounds which are very powerful in their narcotizing properties but yet so readily broken down in the animal organism that anesthesia of only a limited time results. The most popular products in this class are in the barbituric acid series.

At the present time, as will be explained later, the properties of certain compounds in the barbituric acid series are such that one single administration will give anesthesia of only ten to 15 minutes, so replacing, for instance, ethylene chloride, whereas on the other hand other compounds produce anesthesia of one to two hours duration, replacing surgical chloroform or ether anesthesia.

## Action

As you probably know, one of the most accepted theories concerning the action of general anesthetics is the lipoid solubility theory advanced by Overton and Meyer. Experiments show that the barbiturates generally follow this theory to the same extent as volatile anesthetics, so giving evidence that in the bio-physical and bio-chemical events those two groups of anesthetics (volatile and non-volatile) act principally in the same way, the principle being that any form of anesthetic must enter the blood-stream and reach the higher nerve centers, the brain and cord, on which it exerts its specific action. The portal of entry, of course, will have considerable effect on the speed of onset and duration of anesthesia attained.

Since Hewitt has stressed the importance of the action which the anesthetic exerts upon respiration and circulation, and furthermore mentioned that no permanent damage should follow the anesthesia, let us see how those newer barbituric acid derivatives comply with those requests. Pharmacological investigation has shown that, if properly used, some of these compounds compare very favorably with the older volatile anesthetics. Let us refer to two of the short-acting barbiturate groups which compare favorably and will be given most attention in this discussion—nembutal and pentothal sodium.

Nembutal, known chemically as pentobarbital sodium, when given in properly controlled doses, has little effect upon the circulation, causing little if any fall in blood-pressure. This compound, however, does affect respiration, causing a decrease in size and rate of respiratory movements. This is the reason that

administration should always be slow and observation of the patient at all times should guide the dosage.

Pentothal sodium, while a faster and shorter-acting anesthetic, needs even more attention in its administration, since generally barbiturates possessing faster and shorter action are the more depressant upon the respiration. However, even pentothal sodium, when properly administered, does not have a dangerous effect on respiration and, if properly used, has no depressant effect upon the blood-pressure.

Both drugs possess practically no damaging effect upon the animal organism, since both are broken down completely in the liver, with no damaging after effect on that organ. On the other hand, it is clear that an animal suffering from a liver disease will be hampered in the normal destruction of pentothal or nembutal, and the state of health of the patient should always be considered prior to induction of anesthesia. Also, general toxemia, which always is accompanied by the severe disturbance of the metabolism, asks for special caution in the administration of any anesthetic, volatile or non-volatile.

## Dosage

For correct understanding of the dosage of a non-volatile anesthetic, one must make the following consideration. There is no way by which you can arrive at a definite dosage standard of either volatile or non-volatile anesthetics for man or animal, since both have to be adapted to the purpose, as well as to the specific individual reaction of the patient. Both ether and chloroform react differently on various species of animals and the only satisfactory way that either can be used for uniform results is to adapt the amount to the individual requirements of the patient. The same idea must be observed in the administration of a non-volatile anesthetic, for instance, nembutal or pentothal sodium. This means that those figures which are generally given as the dosage for these drugs are average doses resulting from many experiments on different animals, but we should never forget that we do not know how the particular animal which we have to anesthetize ranks in its individual susceptibility to the average dose.

From the wide use of nembutal clinically and the extensive pharmacological work which has been conducted during the past few years, the conclusions are that nembutal must be administered by the intravenous method, to obtain uniform results in all types of cases. The work of Professor Wright, of the Royal Veterinary College, London, advocates this method of administration. Professor Wright has published, on two or three occasions, some

scientific data on nembutal anesthesia in animals. His first paper dealt with the intraperitoneal and oral uses of the drug, in which he was successful in obtaining uniform results in only about 50 per cent of his cases. Continuing his investigational work with nembutal and resorting to the intravenous use of the drug, his results were markedly different. Professor Wright's conclusions, like those of other investigators, are that nembutal must always be given intravenously and stress the importance of slow administration, allowing at least two or three minutes for the time of injection, watching the patient carefully during the assessment of anesthesia to determine the effect of the drug, and injecting only that amount which will abolish reflexes. The dosage, therefore, which has been recommended in the literature and used widely ($\frac{1}{5}$ grain per pound body weight) can be considered only as the average requirement for small animals and cannot be employed routinely with the expectancy of uniform results.

Confirming Professor Wright's experiments, pharmacological investigations have shown that a marked difference exists in the tolerated dose of nembutal in individual animals, this difference being about 25 per cent higher or lower than the average. For example, while the average lethal dose for the rabbit is considered to be 45 mg per kilogram, some individuals tolerate up to 60 mg and others succumb even at 35 mg. These results have been obtained by using a new technic, in which nembutal in a suitable dilution was slowly injected intravenously in the form of a drug infusion until the respiration stopped. Obviously this method gives us the individual tolerated dose for each animal examined. (Details on methods and results will be published later.)

With the wide use of nembutal as a general anesthetic in all ages and classes of animals, varying results have been reported during the past two years. These varying results are usually reported from the intraperitoneal use of the drug; however, in some instances they are reported where nembutal was used intravenously, especially where it was used in a definite dosage of grains per pound body weight and not the amount of drug which would abolish reflexes in the individual animal.

## Stability

There has been a suggestion that nembutal deteriorates and does not maintain its potency. Careful chemical, as well as pharmacological, assays show that nembutal in capsule form is indefinitely stable unless exposed to moisture and that nembutal sterile

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solution also remains stable for long periods of time unless for some reason the solution shows a precipitate.

From the extensive clinical and pharmacological investigations of nembutal, it is concluded that the varying results, therefore, are due to improper dosage, varying tolerance of the individual animal or, in the case of intraperitoneal use of the drug, a faulty technic. Faulty technic probably occurs more often than one would suspect, especially in the intraperitoneal use of nembutal, as in many hospitalized animals they are brought to the operating table with a distended bladder and it is very easy to inject the solution into that organ. It is also possible to inject the solution into a distended gut, and in either instance there will be no absorption or sedative effect upon the animal. Subcutaneous injection of the drug will also result in slow absorption and only mild anesthesia.

One request of Hewitt, namely, that the anesthetic should fade out soon after its administration, is almost entirely fulfilled by pentothal sodium. This drug is, indeed, on one side so powerful in action and on the other side so quickly and completely destroyed in the body that only short anesthesia results.

Pentothal sodium is particularly indicated for operative procedures of short duration—ten to 20 minutes. The drug must always be injected strictly intravenously, since the solution is quite highly alkaline and if introduced outside the vein may cause tissue irritation.

A solution containing one grain in each cubic centimeter is preferred for small-animal use. Unfortunately pentothal sodium is not stable in tablet or capsule form, nor is the solution stable for any period of time. Solutions prepared with chemically pure distilled water will remain effective for six to eight hours, but it is advised that only freshly prepared solutions be employed.

Pentothal sodium is not intended to replace nembutal as a surgical anesthetic. Its application in veterinary practice is particularly advantageous in cases of minor surgery, dentistry, X-ray and other operations of short duration. The dosage of pentothal sodium varies more greatly than that of nembutal. From $\frac{1}{8}$ to $\frac{1}{5}$ grain per pound body weight will cover the dosage range. However $\frac{1}{7}$ to $\frac{1}{6}$ grain is usually employed and will produce anesthesia for a period of ten to 15 minutes.

In the administration of pentothal sodium the required dosage should always be given slowly. A satisfactory procedure is to determine the amount of solution required for the animal, figuring $\frac{1}{7}$ to $\frac{1}{6}$ grain per pound body weight. One-half of the dose

should be injected in a period of one to one and one-half minutes, then, after a 30-second pause, complete the injection until reflexes have been abolished. In many instances reflexes will be abolished without using the entire dose which has been previously calculated.

Pentothal sodium is rapidly destroyed in the liver. However, repeated use of the drug on animals has no bad effect upon the liver tissue or general metabolism.

The one marked advantage of these two barbiturates, nembutal and pentothal, as compared with the volatile anesthetics is the much slighter and shorter state of excitement during the induction of anesthesia. There does, however, appear in certain cases a state of delirium during recovery from nembutal, and to overcome this objectionable feature the combination use of morphine-atropine and nembutal is recommended.

## Technic

The following technic is widely employed in practice and gives uniform results. Administer morphine and atropine, either subcutaneously or intravenously, in a suitable dose, depending on the size of the animal, $\frac{1}{8}$ grain of morphine and $\frac{1}{200}$ grain of atropine for the toy breeds; $\frac{1}{4}$ grain of morphine and $\frac{1}{150}$ grain of atropine for those of medium size, and $\frac{1}{2}$ to 1 grain of morphine and $\frac{1}{100}$ grain of atropine for the larger breeds. Allow at least 30 minutes, and preferably one hour, for the morphine and atropine to take effect, then induce surgical anesthesia with nembutal, giving only the amount of drug intravenously which will abolish reflexes. A profound state of surgical anesthesia will result with recovery free from excitement and delirium.

Barbiturate anesthesia is definitely a step forward in canine anesthesia; nembutal and pentothal sodium definitely possess outstanding properties over the older non-volatile and volatile anesthetics. Although these new barbituric acid compounds are very powerful in action, when introduced into the blood-stream, they can be depended upon to induce just as safe and efficient anesthesia as any of the volatile anesthetics heretofore employed, if care and caution as well as individual susceptibility are observed in each case.

## Discussion

Dr. O. B. Morgan: As practitioners we have learned a lot of things about giving nembutal. We have found it convenient to have a pair of scales in our office with which to weigh a dog. We estimate about 1½ grains in a capsule to each nine pounds of body weight. Some long-haired dogs don't weigh as much as you would think. When the dosage is prepared, perhaps you are not so experienced in introducing

it as you should be, and in that event you are quite apt to give too much anesthetic.

I believe Dr. Sweebe's point was well taken that you should take plenty of time to inject nembutal. I do not believe he mentioned anything about using it in cass of cesarean section. I do not believe the drug should be used in that operation. There is a chance that the pups will be lost. I have been told by others that that is possible.

Nothing, I believe, was said about nembutal in cats. Cats seem to tolerate the drug very well, and seem to be pretty safe. The average cat will take about a grain and a half of nembutal very nicely. It is a rare case that requires a larger dose of the drug for an average-size cat.

## Third United International Congress of Tropical Medicine and Malaria

The Third International Congress of Tropical Medicine and Malaria will be held in Amsterdam, Netherlands, from September 26 to October 1, 1938. A recent announcement indicated that the Netherlands government will invite the governments of other countries to send official delegates. In some countries, national committees for the Congress have been formed. The membership fee in the Congress has been fixed at 1 pound (9 francs, Dutch currency), and all members will receive the printed reports of the Congress. Present plans call for discussions of the following subjects on the program of the Tropical Medicine Section: (1) Deficiency Diseases, (2) Yellow Fever, (3) Leptospirae, and (4) Filariasis. The last mentioned subject will replace Black Water Fever, which was on the program of the 1932 Congress.

The program of the Malaria Section will include the following subjects: (1) Anopheles Varieties, (2) Strains of Parasites, (3) Treatment with Quinine and the New Synthetic Drugs, (4) Prophylaxis, and (5) Sanitation and Colonization.

The Congress also will include a section on Comparative Tropical Pathology for which a tentative program has been arranged to include: (1) Deficiency Diseases, (2) Measures Against Hydrophobia, (3) Rickettsiae, and (4) Extermination of the Tsetse Fly.

## Westminster Kennel Club Show

The 62nd annual show of the Westminster Kennel Club will be held at Madison Square Garden, New York City, February 10-11-12. It is expected that the show this year will attract 3,000 entries. As a matter of fact, the entries will be limited to that number in order that all dogs may be benched comfortably.

# MOLDY CORN POISONING IN HORSES*

*By* L. H. SCHWARTE, *Ames, Iowa*
*Veterinary Research Institute, Iowa State College*

Most of the veterinarians who are located in sections of the country in which corn is extensively raised are more or less familiar with moldy corn poisoning. They realize the serious losses which have resulted from time to time due to the feeding of toxic corn to horses. This condition has been recognized for years, and reports published from time to time indicate that losses from this cause are by no means limited to one particular locality. Time does not permit us to review the literature on this subject, but it might be of interest to recall the report of Buckley and MacCallum,[1] in 1901, who associated a pathologic condition in horses with the feeding of moldy fodder. Butler[2] published the results of his feeding experiment the following year, by which he produced a typical case of the non-virus disease causing gross liquefaction necrosis in the brain of the horse which was fed moldy ear corn. This early experimental work of Butler definitely showed that there were several separate conditions which were responsible for central nervous disturbances in the horse and that one of these, associated with degeneration and liquefaction necrosis in the brain, was caused by toxic corn.

In presenting this subject, a brief review of some of our studies, including the field cases and the results of our experimental work, will be made. An attempt will be made to differentiate this condition from some other diseases of horses in which the central nervous system is involved, resulting in clinical symptoms which are similar and in some cases identical to moldy corn poisoning. There has been considerable confusion developing in the minds of the practitioners recently regarding certain factors and pathologic changes which make it possible to differentiate some of these conditions. This confusion may be the result of some recent publications.

## FIELD CASES

For the past two years, during the winter and spring, many cases have been brought to our attention which manifested symptoms indicating a severe central nervous disorder. The majority of these cases were diagnosed clinically as the specific virus encephalomyelitis. Examination of the brains of these horses showed no evidence of the virus disease. Microscopic examination

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failed to reveal any pathologic changes characteristic of the virus disease. Furthermore, all attempts to demonstrate the presence of virus by experimental inoculation of animals failed. The brain tissue of these horses invariably showed various degrees of degeneration and liquefaction necrosis, which were confined to the white substance of the cerebral hemispheres. One horse which showed definite clinical symptoms was destroyed. The examination of the entire brain of this animal showed only a small edematous area about one inch in diameter in the base of the brain. This small lesion was of a yellowish-orange color. Numerous other cases reported by veterinarians throughout the state occur-

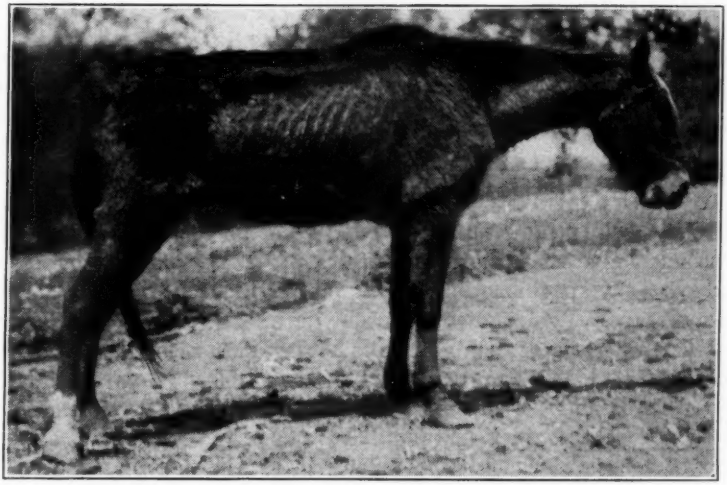

FIG. 1. Experimental moldy corn poisoning in horse showing depression and marked incoördination. Note position of hind legs. (Schwarte, Biester and Murray.)

ring at this time of the year, showing the same clinical symptoms, revealed similar pathologic changes in the brain tissue.

## EXPERIMENTAL

A carefully controlled experiment was carried out in an effort to reproduce this condition by feeding moldy corn of known toxicity. Corn was secured from a farm located in a region where heavy losses were reported during the month of November. This corn was purchased on March 30 and appeared to be in much better condition than it appeared when fed to horses the previous November.

Five horses were placed in a dry lot and fed moldy corn and a good grade of corn fodder. Three horses used as controls were

fed clean corn and corn fodder. All of the horses fed moldy corn developed well defined clinical symptoms. Those which died showed extensive brain lesions, namely, edema, hemorrhage and liquefaction necrosis in the white brain matter. The majority of these necrotic areas were yellowish-orange in color and of a creamy consistency. The control horses which were fed clean corn and corn fodder failed to develop any clinical symptoms or any untoward response. They were released in good condition after the close of the experiment.

Bacterial cultures made from the brain tissue of the horses fed experimentally which either died or were destroyed after showing well defined symptoms, were negative. The brain tissues sent to the laboratory from field cases, which arrived in good condition, likewise proved to be free from bacterial infection. Intracranial inoculation of laboratory animals in all instances failed to show any indication of virus infection.

## Comparison with Other Conditions Which Frequently Resemble Moldy Corn Poisoning

The difficulty of selecting suitable names, by which we may instantaneously associate a certain definite pathologic condition caused by a specific etiological factor, has been recognized for many years. The condition now being discussed has been designated tentatively as moldy corn poisoning in horses. This at once differentiates it from the specific virus encephalomyelitis and other conditions which may be found to produce symptoms characteristic of a central nervous disturbance. It seems that equine encephalomyelitis should be reserved for the specific virus disease because it is quite comparable to the specific virus disease in man known as poliomyelitis. We know from our experience with the various species of domesticated animals that certain viruses, bacterial infections and specific toxins may cause an encephalitis with well defined clinical symptoms characteristic of a central nervous disturbance. Consequently moldy corn poisoning in horses has been confused with the specific virus encephalomyelitis and botulism.

The seasonal occurrence and the history of the case may be guiding factors to the practitioner in making a differential diagnosis. As you will recall, moldy corn poisoning usually appears in Iowa in November and continues through the winter months, even as late as May, during which time the virus disease has subsided. It is possible, however, to have an overlapping in the occurrence of these two diseases in October and November, depend-

ing on the temperature and rainfall affecting the development of the corn. The specific virus encephalomyelitis is seldom encountered later than two weeks following the first killing frost. In moldy corn poisoning, the symptoms manifested depend on the extent and location of the lesions in the brain.

The course of the disease depends on the toxicity of the corn, the quantity of toxic corn consumed and the tolerance of the individual to the toxin. Again the location and extent of the lesions may definitely influence the course of the disease. The pathologic changes associated with moldy corn poisoning include necrosis, edema, definite hemorrhage and degenerative processes. On the other hand, the pathologic changes associated with the specific virus encephalomyelitis are perivascular infiltration, focal infiltration, and invasion and destruction of the nerve cells by the leukocytes. Bronzing of the nerve cells is often observed. Considering the seasonal occurrence and history of the case with the pathologic changes described, it is possible to differentiate the virus disease from moldy corn poisoning.

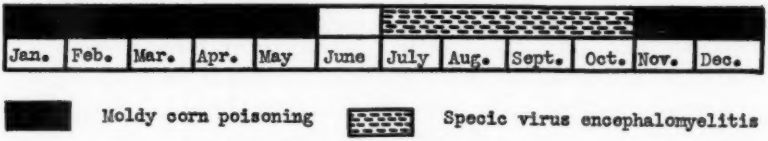


FIG. 2. Chart showing seasonal occurrence of moldy corn poisoning and encephalomyelitis. (Schwarte, Biester and Murray.)

In cases where *Clostridium botulinum* or its toxin is suspected as the cause of a pathologic condition in horses, other farm animals which have access to this feed may also be affected. A positive diagnosis of botulinus poisoning can be made only when the toxin is found to be present in the feed. The therapeutic administration of botulinus antitoxin usually relieves the condition. Prophylactic treatment with antitoxin will protect animals. *Cl. botulinum* or its toxin has never been known to produce lesions similar to those caused by moldy corn poisoning or the specific virus encephalomyelitis.

## DISCUSSION

Encephalitis in itself is a rather general term indicating an inflammation of the brain regardless of the cause. It is not necessary to repeat that which has already been said regarding the various conditions resulting in an inflammatory reaction in the brain. The term "infectious type of the disease," which was used in a recently published report, can refer only to the specific virus

encephalomyelitis. This case was reported to have occurred during the winter months, but the presence of the specific virus was not demonstrated. Conditions caused by moldy corn or other toxic agents can hardly be considered of an infectious nature. The lesions and histologic findings described in this report do not conform to those recognized as being caused by the specific virus encephalomyelitis, based on carefully controlled experimental work. To say nothing of the work done by members of our own staff, both in the field and in the laboratory, in connection with the specific virus encephalomyelitis and other closely related conditions, we must accept the results reported by such men as Records and Vawter, Shahan and Giltner, and Dr. K. F. Meyer and his associates. These investigators not only isolated the specific viruses and typed them but also worked out the mode of transmission, the characteristics of the disease, the pathologic and histologic changes associated with it, and the variations found in the different types of virus. This work was based on carefully controlled experimental work. These workers have never reported liquefaction necrosis, degenerative changes or extensive hemorrhage in their investigations with the specific virus encephalomyelitis. It does not seem possible that men of their experience and training in years of careful work would fail to observe such tissue changes in their histologic studies. We cannot accept theoretical deductions based on an isolated field case in place of the evidence reported by such recognized authorities based on the results of controlled experiments.

I do not want to be misunderstood regarding the value of case reports and field experiences. We should have many more of these reports published. They are of great interest and value to the practitioner, clinician and research worker as well. They furnish much information concerning the occurrence, frequency and complications, together with practical and effective means of handling these cases in the field. Such reports furnish information and material for research work. However, these cases must be presented in an accurate and concise form and not in such a way as to conflict with the established and accepted scientific data obtained through carefully controlled experimental work. This is not so easy and simple as it may seem, because of the vast number of complications which are observed in the field; as for instance the complications that may arise from cases of moldy corn poisoning and the specific virus encephalomyelitis in horses.

We do know that a certain number of horses do recover from both conditions. We do know that definite tissue changes have

taken place in order to cause definite clinical symptoms. We do not know, however, to what extent these changes may progress and yet have the animal recover. We do not know to what extent or how complete the regeneration of these tissues may be following recovery. It would be possible to find lesions typical of a specific disease in a recovered animal which died later from an entirely different cause. We always have to keep in mind the possibilities of complications which might occur and which frequently do occur in field cases.

## Prevention and Treatment

The practitioner is often called upon to treat horses suffering from moldy corn poisoning. There is no specific therapeutic agent known up to the present time which is effective in relieving this condition. The horses should be removed from the field, or, if fed in the barn, should not have access to toxic feed. A laxative is indicated to eliminate the toxic material ingested. A light feed of bran and good hay should be continued for some time after improvement is observed. The animal should have access to fresh water at all times. In advanced cases, the removal of the stomach contents is indicated. Water may be administered as indicated by means of a stomach-tube until the animal is able to drink. In order to prevent losses from this cause it is necessary to eliminate the possible consumption of toxic corn. Some clients who have experienced losses from this condition no longer allow their horses to run in the cornfield. They also provide muzzles for their horses in the field during the harvesting of their corn. These clients have had no further trouble.

## Summary

A brief review of moldy corn poisoning in horses, including field cases and the reproduction of this condition by carefully controlled experimental studies, has been presented. The seasonal occurrence in Iowa, the typical lesions and pathologic tissue changes, making it possible to differentiate moldy corn poisoning from other conditions manifesting similar clinical symptoms, have been described. Suitable measures for the prevention and treatment of this condition which have proved to be the most effective have been recommended.

## References

[1]Buckley, S. S., and MacCallum, W. G.: Acute hemorrhagic encephalitis prevalent among horses in Maryland. Amer. Vet. Rev., xxv (1901), pp. 99-102.

[2]Butler, T.: Notes on a feeding experiment to produce leucoencephalitis in horses with positive results. Amer. Vet. Rev., xxvi (1902), pp. 748-751.

DISCUSSION

DR. N. S. MAYO: I recall this disease in Kansas, in 1890 and 1891, when I made investigations and published the results. I found the same conditions that Dr. Schwarte has described. Cases in which recovery took place were exceedingly rare. The disease, as observed at that time, became active early in the fall, in October, and disappeared in March. The disease was associated with damage done by the green-corn worm. The damaged corn became infested with mould. Almost invariably it was the horse walking next to the corn row that bit off tips of the ears that contracted the disease. The farmer got so that he would take the shelled corn, soak it in buckets of water and skim off the moulded kernels. A very common symptom of this disease was paralysis of the muscles. I do not recall a case that lived more than two or three days. It was essentially a seasonal disease. It appeared in years when the corn was attacked by the green-corn worm.

DR. G. L. DUNLAP: Have you been able to isolate the toxic substance from the corn?

DR. L. H. SCHWARTE: We are trying to isolate the organism responsible for the disease.

DR. L. W. GOSS: In 1907, we did considerable work with the disease, but we published practically nothing. We continuously had hopes of having something to publish rather than negative results. In our experience, we found nothing with which we could reproduce the disease.

DR. T. E. WILKE: In southeastern Missouri, a good many years ago, there was a veterinarian who treated this disease by injecting a strong caustic under the skin. When I first went down there, he gave me a lot of trouble. The community attributed every recovery to this treatment. In the majority of cases where this disease occurred, the drainage was poor. Since then, some of the farms have been well drained.

## Bureau Transfers

DR. BURNETT C. JOHNSON (U. S. C. V. S. '20), from Milwaukee, Wis., to South Saint Joseph, Mo., in charge of meat inspection.

DR. JOHN A. PATTON (St. Jos. '14), from Ottumwa, Iowa, to Milwaukee, Wis., in charge of meat inspection.

DR. GLEN H. RANDALL (K. C. V. C.. '11), from Kansas City, Kan., to Moultrie, Ga., on meat inspection.

DR. F. W. CRAWFORD (K. S. C. '23), from Sioux Falls, S. Dak., to Kansas City, Kan., as assistant inspector-in-charge of meat inspection.

DR. DONALD R. APPEL (Cin. '20), from Montgomery, Ala., to Oklahoma City, Okla., on meat inspection.

DR. ROBERT C. DICKSON (K. C. V. C. '18), from Little Rock, Ark., to Kansas City, Kan., on meat inspection.

DR. LEO V. HARDY (St. Jos. '20), from Fort Dodge, Iowa, to Newark, N. J., in charge of meat inspection.

DR. JAMES C. MELVIN (A. P. I. '26), from Nashville, Tenn., to Saint Louis, Mo., on meat inspection.

DR. DWIGHT L. CECIL (McK. '17), from Newark, N. J., to Chicago, Ill., on meat inspection.

DR. ROBERT L. DURR (A. P. I. '32), from Atlanta, Ga., to Montgomery, Ala., on Bang's disease and tuberculosis eradication.

DR. ALBERT D. GLOVER, JR. (Colo. '36), from New York, N. Y., to Saint Louis, Mo., on meat inspection.

DR. AUSTIN J. MURPHY (Chi. '15), from South Saint Paul, Minn., to San Diego, on meat inspection.

# FURTHER OBSERVATIONS ON OAT HAY POISONING*

*By* FRANK THORP, JR., *Fort Collins, Colo.*
*Colorado Agricultural Experiment Station*

Losses of cattle following consumption of oat hay were reported by Newsom *et al*[1] from this laboratory. Data were presented on the experimental feeding of oat hay to cattle and sheep, and the hay and hay extracts to rabbits and guinea pigs. The symptoms were suggestive of hydrocyanic acid poisoning and sodium picrate paper tests, positive for this acid, were obtained from the rumen contents of some of the experimentally killed cattle but not in the hay.

Rogers and Boyd[2] mentioned death among cattle being fed oat hay containing Canadian thistles. They were unable to demonstrate hydrocyanic acid in the hay.

Wilcox[3] attributed the loss of twelve dairy cattle in Montana to the feeding of smutty oat hay. The hay was cut green, stored and fed in the winter. The animals died during a period of twelve hours after eating and showed frenzy. On autopsy a gastritis was present.

Although entitled "Oat Hay Poisoning," this paper will include cases of poisoning by hays other than oat since the circumstances appear similar.

## OUTBREAKS SINCE 1936

A rancher near Haxtun, Colorado, reported the loss of six cattle on April 5, 1937, after feeding wheat straw from the butt of a stack. On March 31, a light rain had fallen. Pool water was available for the cattle to drink.

On a ranch near Gulnare, Colorado, eight cattle out of a herd of 57 died during a period of three to 3½ hours after eating oat hay containing pig weed. The hay was fed during a snow storm. Two cows aborted their calves shortly after this outbreak. The remainder of the stack was fed to cattle during the next twelve days without loss. A second stack of the same kind of hay was fed to horses without loss. From a third stack which was wet on top, two loads of wet hay were fed sparingly to horses and cattle in a barn without loss. However, on April 24, the 49 remaining cattle were fed dry oat hay from this stack and twelve

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died in about three to 3½ hours. Water was available at all times.

Dr. L. Van Es[4] described cattle losses on a farm near Bartley, Nebraska, during the first week in April of 1937. Oat hay from the bottom of a stack that had been rained upon the previous day was fed to some yearling heifers in a dry lot and to 85 cows in a pasture where spring water was available. The hay was fed about 4:00 p. m. and by the next morning 24 cows had died. The heifers, which had had no water, were not affected. About four days later, six cows aborted their calves.

## Outbreaks Investigated Experimentally

*Ranch I:* On the same ranch where 67 cattle had died in 1936, after eating oat hay (Newsom *et al*[1]), eleven cattle out of about 50 died March 26, 1937, after consuming, during a snow storm, hay consisting largely of pig weeds, but also containing Russian thistle, immature barley and native hay. It had been cut when immature, having acted as a nurse crop for new alfalfa seeding. These cattle had been fed cane hay all winter until the last five feedings when the above hay was given. The hay was taken from the bottom of the stack, and was damp and moldy. It was hauled once a day onto the ground in order to make the animals forage for grass. The affected cattle died within 2½ hours after eating, and of the survivors six or seven aborted their calves within three days.

After the cattle had been dead about 20 hours, sodium picrate papers (Henrici[5]) were inserted into the rumens of four of the animals using the technic described by Newsom *et al.*[1] No hydrocyanic acid was detected after nearly two hours.* This may have been due to the freezing temperature, since the test works best at 30° C. or higher.

Some of the hay was brought to the laboratory about 24 hours after death of the cattle. A fruit jar was immediately filled, sealed and set in the refrigerator. Two days later, it tested negative for hydrocyanic acid and arsenic, according to Professor J. W. Tobiska, chemist for the Experiment Station. A portion of the hay was cut into pieces about one inch long, and placed in quart fruit jars, which were then filled with tap water, sealed and set in the refrigerator over night for extraction. This aqueous extract was then given *per os* to the following animals starved about 24 hours: Approximately 90 cc to a three-fourths grown lamb, about 1 cc to a guinea pig, and approximately 2 cc to a

*Throughout this paper, unless otherwise stated, this is the method used for the detection of hydrocyanic acid.

rabbit. No symptoms developed, and all appeared normal at the end of 24 hours.

About 50 cc of the aqueous extract was filtered through a Seitz clarifying disc and then through a Seitz (EK) filter. The filtrate was cultured aerobically and anaerobically and found to be sterile after three days at 37° C. Five cc was injected intraperitoneally into a starved guinea pig. A few seconds later, slight abdominal distress was noted, but after a minute no further symptoms developed. A starved rabbit was injected intraperitoneally with 10 cc of the sterile filtrate. Neither animal showed symptoms during the next 24 hours.

Two days after the outbreak, and continuing for four days, portions of the hay were wetted and fed to a ewe. A lamb was fed in the same way for three days. The sheep were apparently normal 48 hours after the last feeding.

The remainder of the stack of hay was fed to horses without any ill effect. Mr. E. H. Grant, owner of the ranch, was informed that a specimen of the hay in question submitted to the Bureau of Animal Industry showed a trace of hydrocyanic acid.

*Ranch II:* A rancher near Fort Collins, Colorado, fed 30 head of cattle on March 26, during a snow storm, some oat hay from the top of a stack. This was the first feeding from this stack. Five died during the night. That same evening a limited amount of dry oat hay was fed to the dairy cattle, and no ill effects were observed. Since, during the previous day, they had run on pasture infested with larkspur, the rancher's neighbors convinced him that this weed was the cause of his losses. About ten days later, it was found, however, that the larkspur was not sufficiently grown to cause trouble. On April 7, the remaining 25 cattle were fed oat hay from the top of the stack in the afternoon during a snow storm. That evening six cattle became sick, and three of these died. They were observed to be sick about three to four hours after feeding. The animals acted as though fatigued and showed incoördination of gait, which became more pronounced on driving and during excitement. The temperature was normal, respiration increased and heart-beat rapid and audible. The mucous membranes were somewhat cyanotic, and the blood was very dark.

The two sickest animals were each given 5 grams of sodium nitrite and 7.5 grams of sodium thiosulfate intravenously in a 30 per cent aqueous solution. All three sick animals recovered.* That same evening, dry oat hay below the top of the stack was

*Dr. James Farquharson and Dr. I. E. Newsom administered the treatment, and reported the clinical symptoms.

fed to dairy cattle, and no trouble followed. This same hay from all parts of the stack was then fed to horses for about a month with no ill effects.

At the beginning of the feeding period in early winter, the cattle had been fed a stack of fairly mature oat hay. Following this they were given corn fodder and cane fodder, and then the oat hay in question. The latter had been cut while in the milk and light dough stage. It was well cured and stacked outside. There were very few pig weeds, lambs quarters, Russian thistles or sunflowers present. The Botany Department made the identification of the weeds, and stated that no poisonous plants could be found in the hay. The top of the stack was discolored, moldy and damp, while the remainder was dry and green and appeared to be in good condition. It was the opinion of the Station botanists and agronomists alike that the dry hay would be considered of excellent quality.

On the morning following death of the three cattle, the sodium picrate paper test for hydrocyanic acid was run on the rumen contents. The morning was cold. All tests were negative.

*Investigation of hay:* Some of the moldy oat hay was brought to the laboratory, and was reported by the Station chemist to be negative for hydrocyanic acid. A cow that had been starved for 24 hours was fed this hay, wetted, for three days with no ill effects. The cow was then given by stomach-tube two gallons of a 24-hour aqueous extract of the hay, one gallon into the rumen and one gallon into the reticulum, with no ill effects in the next 24 hours. The extract was prepared by soaking five gallons of the moldy oat hay in tap water at a temperature of about 18°C. The following day, two gallons of a 45-minute aqueous extract (prepared as the 24-hour extract) was given to the cow *via* stomach-tube into the rumen. The animal remained normal.

On April 12, in another sample of the moldy oat hay the weeds were separated from the oats. The Station chemist reported a slight trace of hydrocyanic acid in the oat hay, but no selenium, while the weeds were negative for both.

*Feeding experiment:* Six young cows of mixed breeding were donated for further experimentation with the oat hay on ranch II.* On May 12, they were placed in a dry corral surrounded by a dry lot on ranch II, and were given no food or water. This same day, in order to simulate conditions occurring during the natural outbreak, the top of the stack that had caused the previous losses

*We are greatly indebted to Mr. E. H. Grant, of Denver, owner of ranch I, for this generous gift, and to Mr. Sam Thompson, owner of ranch II, for furnishing oat hay and accommodations for the cattle.

was wetted twice with water. On May 13, the cattle were divided into two groups of three each. One group was fed good, dry oat hay from the middle of the stack. It was well wetted before and during the feeding. Water was given after feeding. The other group was fed the moldy, damp hay from the top of the stack. The portion fed was further wetted just before feeding. Two of the animals ate well, but the third did not, due to the presence of some grass in the lot. The experiment was continued in the same way on May 14, except that the moldy hay group was confined to a dry lot and both groups were watered while eating, after eating, and again during the evening. All animals remained normal.

On the morning of May 15, the cattle were put together and fed moldy hay which was sprinkled just before feeding. The hay was placed in a stock tank, some excess water being present in the bottom of the tank. The animals were watered at noon and again in the evening. They remained apparently normal. The same procedure was used again on the fourth day, May 16, at 8:15 a. m. All of the cattle had become accustomed to the feed by this time, and were hungry. One of the animals (cow 5), being a meek individual, was fed by herself.

When seen at 4:15 p. m., animals 1 and 2 had just died, and animal 3 was down. At 4:38 p. m., the latter was given 4 grams of sodium nitrite in a 20 per cent solution intravenously and, no improvement being noted, she was then given at 4:50 p. m. 7 grams of sodium thiosulfate in a 10 per cent solution intravenously. The animal died five minutes later. At 6:00 p. m., the three surviving animals were examined clinically, and no cyanosis was observed. At 5:00 p. m., sodium picrate papers were inserted into the rumens of the three dead cattle. About 5:10 p. m., a small sample of the rumen contents of each of the three animals was removed, and the sodium picrate paper test applied, the tubes being placed in hot water. In 25 minutes a positive test for hydrocyanic acid was given by the rumen contents of animal 3, but not by animals 1 and 2. At 5:45 p. m., another sample of rumen contents from animal 3 was tested, but was negative at 6:55 p. m., and remained so, as did the other rumen samples after a period of 96 hours at 37°C. after transportation to the laboratory. The sodium picrate papers which had been placed in the rumens were negative for hydrocyanic acid at 8:30 p. m., and also at 8:30 a. m. the following day. The paper placed in the rumen of animal 3 was touched by the fermenting contents during the night, and the test was, therefore, unsatisfactory. At 7:30 p. m. the same day as the other casualties, animal 4 was

noticed to be sick. At 8:05 p. m., the animal was down and was given 6 grams of sodium nitrite in a 30 per cent solution intravenously. This was immediately followed by 5 grams of sodium thiosulfate in a 10 per cent solution, intravenously. The animal died about 8:15 p. m. A small sample of the rumen contents was immediately removed and the sodium picrate paper test applied, the tube being placed in the automobile radiator for 22 minutes and then transported to the laboratory, and set at 37° C. No trace of hydrocyanic acid was detected after a period of 96 hours. A sodium picrate paper placed in the rumen at 8:20 p. m., May 16, showed a trace of hydrocyanic acid twelve hours later.

On May 17, the two remaining animals (5 and 6) were watered just before feeding and in place of the mouldy hay previously fed were given, at 8:35 a. m., good, dry oat hay taken from the middle of the stack. Animal 5 was noticed to be sick at 2:15 p. m. Upon our arrival at the ranch at 3:15 p. m., the cow was found to be down and almost dead. Four grams of sodium nitrite in a 10 per cent solution was given at once intravenously, followed by 7 grams of a 10 per cent solution of sodium thiosulfate intravenously. The cow died seven minutes later. Some of the rumen contents was collected as it ran from the nostrils, and was corked and taken immediately to the laboratory. A sodium picrate paper test run on the sample at 37°C. was positive for hydrocyanic acid the next morning. The cow was allowed to lie over night before attempting a direct hydrocyanic acid test on the rumen contents. At 7:15 a. m., May 18, the rumen was tapped and the gas allowed to flow past the sodium picrate paper. The paper was then removed long enough to obtain a sample of rumen contents, was reinserted and, at 10:30 a. m., gave a positive test for hydrocyanic acid. The rumen contents, when tested at the laboratory, were negative for hydrocyanic acid.

Animal 6 was fed the dry oat hay from May 18 until May 25, being watered before each feeding. On the latter date, it rained hard and the cow was given neither feed nor water. On May 26, at 1:30 p. m., the animal was fed the good oat hay, which was damp from the rain of the previous day. Water was given just before feeding. The cow was apparently normal at 6:45 p. m., but about 20 minutes later was down and within a minute or so was dead. At 7:40 p. m., some of the rumen contents was taken as it ran from the animal's nostrils, and a sample of blood was drawn. Both were sealed and tested at the laboratory for hydrocyanic acid. Both were negative after 96 hours at 37°C.

The animal was allowed to lie overnight, and at 7:15 a. m., May 27, the rumen was tapped and gas allowed to flow past

sodium picrate papers. After the gas had escaped, some of the rumen contents was removed and sealed and, when tested later at the laboratory for hydrocyanic acid, was negative after 96 hours at 37°C. Sodium picrate papers were reinserted into the rumen, one tube directed dorsally and the other ventrally, the opening around the tubes being packed with gauze. At 10:30 a. m., the paper occupying the ventral position was strongly positive for hydrocyanic acid, while the paper in the dorsal position was slightly positive (table I).

## Symptoms

The author was not present while the animals were standing, the symptoms recorded being observed after the cattle were down. These symptoms were quite uniform in character. The animals made no effort to get up when approached nor could they be induced to rise. The visible mucous membranes were cyanotic, and the blood was a dark chocolate color. The heart-beat was rapid and respiration increased. Just before death, there was a labored breath or two accompanied by a brief easy swing of the feet and head. Those animals that died unobserved showed evidence of having swung the feet and head. Respiration then ceased, the body quivered, the head was drawn back, and the heart continued to beat for a time.

## Postmortem Examination

Two of the animals were autopsied after an interval of one night.

*Animal 5:* Skeletal and heart muscles were of light, cherry red color. On the epicardium there were petechial hemorrhages and the pericardial fluid was tinged with blood. The vascular blood was dark and had clotted. Upon removal of the papillae from the wall of the anterior cul de sac of the rumen, acute congestion was noted. The mucous membrane of the abomasum was uniformly congested throughout. The medulla of the kidney was of a pinkish red color.

*Animal 6:* Postmortem findings were similar to those of animal 5, with the following exceptions, which are in reality exaggerations: hemorrhage in the intercostal muscles over the ninth rib, acute congestion in all parts of the rumen except the dorsal wall, presence of a catarrhal exudate in the abomasum, ecchymotic hemorrhages on the spleen, and pink areas on the cortex of the kidneys.

## Further Experiments

About 1½ gallons of the rumen contents was removed from animal 6, sealed and brought to the laboratory. The liquid was

TABLE I—*Oat-hay feeding experiment.*

| CATTLE | DATE | FEED | WATER | DEATHS | TIME OF DEATH | HCN TEST ON RUMEN CONTENTS* |
|---|---|---|---|---|---|---|
| 3 | 5–13–37 | Good hay, wetted | After eating | 0 | | 0 |
| | 5–14–37 | Good hay, wetted | During and after eating and in evening | 0 | | 0 |
| 3 | 5–13–37 | Moldy hay, wetted | Before and after eating | 0 | | 0 |
| | 5–14–37 | Moldy hay, wetted | During and after eating and in evening | 0 | | 0 |
| 6† | 5–15–37 | Moldy hay, wetted | After eating and in evening | 0 | | 0 |
| | 5–16–37 | Moldy hay, wetted, 8:15 a.m. | None | 4 | 4:15 p.m. to 8:15 p.m. | 1†<br>1 trace<br>2— |
| 2‡ | 5–17–37 | Good hay, dry, 8:35 a.m. | Before eating | 1 | 3:22 p.m. | † |
| 1§ | 5–18–37 to 5–25–37 | Good hay, dry | Before eating | 0 | | 0 |
| | 5–25–37 | None | None | 0 | | 0 |
| | 5–26–37 | Good hay, damp from rain previous day, 1:30 p.m. | Before eating | 1 | 7:00 p.m. | † |

*Solium picrate paper test for hydrocyanic acid.
†Two previous groups combined.
‡The two survivors.
§The one survivor.

expressed, and immediately given *per os* to two rabbits (starved 24 hours) in 10-cc amounts and to two guinea pigs (starved 24 hours) in 5-cc amounts. There were no harmful effects.

The same day some of the moldy wet oat hay was cut into small pieces, put into a fruit jar, covered with tap water and set at 37°C. for 4½ hours. The extract was then filtered off and given *per os* to two rabbits and two guinea pigs, all previously starved, in 10- and 5-cc amounts, respectively. The animals remained apparently normal.

On May 29, a quantity of the wet moldy hay from the top of the stack at ranch II was brought to the laboratory, passed through a meat-chopper and well mixed.* The moisture content was found to be 200 per cent on the dry basis. In an effort to extract the lethal substance from the hay, four samples of 500 grams each were placed in four flasks and the following solvents in 1,500-cc amounts were added, one to each flask: tap water, 95 per cent alcohol, a 1 per cent solution (by weight) of sulfuric acid and a 1 per cent solution of sodium carbonate. The flasks were then corked and from each cork was suspended a moistened piece of sodium picrate paper. The flasks were left at room temperature for 45 to 46 hours. At that time, the paper tests were negative for hydrocyanic acid. The procedure for preparing the different extracts was as follows: The supernatant liquid was poured off and the remaining liquid was expressed by means of a lard-press. The alcoholic extract was allowed to evaporate for ten days at room temperature to a volume of 14 cc and just before administering to rabbits 30 cc of distilled water was added. The aqueous extract was immediately put into suction flasks under a vacuum of about 40 cm of mercury at a temperature of 45 to 50°C., and evaporated to about 60 cc. This procedure took about eleven days. The acid and alkaline extracts were each neutralized, and then centrifuged. The supernatant fluid was decanted, and then evaporated in the same manner as the aqueous extract. Each of the concentrated extracts was divided into two equal parts and given by stomach-tube to two starved rabbits. No ill effects were produced.

Two sheep confined in a dry lot were fed a mixture of good oat hay and moldy oat hay for a period of 16 days. No losses were encountered.

Sixteen samples of oat hay were taken from the stack at ranch II—from the top, middle and bottom. Weeds also were picked out of the hay from the top and bottom of the stack. These sam-

*This part of the investigation was carried out by Dr. H. W. Reuszer, associate bacteriologist, Colorado Agricultural Experiment Station.

ples and also samples of cane hay as positive controls were tested for hydrocyanic acid. After incubation at 37°C. for 48 hours, the results were negative for the oat hay and weeds and positive for the cane.

A sample of the good dry oat hay was tested for hydrocyanic acid by the colorimetric method[6] and found to be negative.*

## Discussion

In a previous publication[1] from the Colorado Agricultural Experiment Station, oat hay was definitely shown to be responsible for deaths among cattle. Investigation of two more recent outbreaks in cattle point to oat hay and weedy barley hay, respectively, as the causes of deaths. Of three outbreaks in cattle reported to us, wheat straw was responsible for one outbreak, damp and dry oat hay for the second, and damp oat hay from the butt of a stack for the third.

Further evidence of the lethal effect of the oat hay responsible for one outbreak was demonstrated experimentally by feeding six cattle the hay in question which with water constituted the ration. All succumbed in from four to 14 days. This confirms the results of Newsom *et al.*[1] The symptoms observed in the experiment animals were very similar to those shown by animals in the original outbreak on the same hay. The syndrome also closely resembled that for hydrocyanic acid poisoning described in the literature.[7] The significance of the positive hydrocyanic acid tests on rumen contents in four of the six animals cannot be definitely stated. Perhaps the two other animals would have given a positive test had they been allowed to remain overnight before opening the rumen. In only two instances was a trace of hydrocyanic reported from the hay in question—once from oat hay (ranch II) and once from weedy barley hay (ranch I). Many other hydrocyanic acid tests were made on the oat hay from ranch II, but they were all negative.

Although in most cases wetness or dampness of the hay seemed an important factor, some evidence is presented to show that death may occur when dry oat hay is fed.

The occurrence of abortion among some of the pregnant animals surviving after eating, the hay was again recorded.

Attempts to kill rabbits and guinea pigs by the oral administration of liquid expressed from the rumen contents of one dead cow were unsuccessful.

*This analysis was made by Mr. Otto H. Coleman, assistant agronomist, Colorado State College.

Aqueous, alcoholic, acid and alkaline extracts of the moldy oat hay proved non-lethal when administered orally to rabbits.

Although the symptoms simulate those of hydrocyanic poisoning and a trace of this substance was found once in the incriminated hay in each of the two investigated outbreaks, proof is far from satisfactory as to the etiology of this condition. The inability to demonstrate hydrocyanic acid in the rumen contents of all animals dead of the disease and in each sample of the offending hay leaves the problem still open for further investigation.

Whatever the etiological agent may be it is quite elusive but rapid in its action. The ultimate solution of the problem will no doubt come from the combined efforts of bio-chemists, botanists and animal pathologists.

## SUMMARY

Serious losses among cattle after the feeding of oat hay, weedy barley hay and wheat straw are described. In all cases but two the hay was fed when damp. Symptoms observed in the cattle are suggestive of hydrocyanic acid poisoning, but in only two instances was a trace of this substance found in the hay. Numerous other hydrocyanic acid tests on the hay were negative.

When the oat hay responsible for one outbreak was fed experimentally to six cattle, all died showing symptoms and lesions typical of oat hay poisoning. The rumen contents of four of the six animals showed positive tests for hydrocyanic acid by the sodium picrate paper method.

Sheep and horses were not injured by feeding the oat hay or weedy barley hay that had proven lethal to cattle.

Aqueous, alcoholic, acid and alkaline extracts of the offending oat hay were harmless when administered to laboratory animals.

## ACKNOWLEDGMENT

The author desires to express his appreciation for the assistance and advice given during the investigation by the following: Dr. I. E. Newsom, Dr. E. N. Stout, Dr. A. W. Deem, Dr. H. W. Reuszer, Dr. James Farquharson, Dr. H. W. Johnson, Dr. L. W. Durrell, Professor J. W. Tobiska and Mr. Otto H. Coleman.

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## Three New Regional Laboratories

Under the provisions of the Bankhead-Jones Act, three new regional laboratories for the investigation of regional problems in agriculture were approved during the fiscal year ended June 30, 1937. The new regional laboratories include one for the study of animal parasites in the Southeast, one for the development of swine breeding in the North Central states, and one for sheep improvement in the range states. These three new laboratories supplement three similar developments of the previous year: the vegetable breeding laboratory for the Southeast, the soybean laboratory in the Corn Belt, and the pasture improvement laboratory for the Northeast. These three projects are now under way.

The laboratory for the study of diseases and parasites of domestic animals is being established at the Alabama Polytechnic Institute, at Auburn. The experiment stations of 13 southeastern states are coöperating with the U. S. Department of Agriculture in the development of the program for regional studies, which will be under way in all the states with headquarters and coördinating direction at Auburn. The states are: Virginia, North Carolina, South Carolina, Georgia, Florida, Alabama, Mississippi, Louisiana, Texas, Arkansas, Tennessee, Kentucky and Oklahoma.

Regional research has been made possible by appropriations under the Bankhead-Jones Act, which, unlike previous legislation for the support of experiment stations, requires state appropriations to offset the federal contributions. Each state complied with this requirement and, on the average, state appropriations were several times the amount required under this provision. Total funds from state sources not only met the new offset requirement, but amounted to about $12,000,000.00, or slightly more than $2.00 from state sources for each $1.00 from federal grants for the support of experiment stations and approximately six times the federal grant offset requirements. Under the Hatch, Adams and Purnell acts, each state continues to receive equal appropriations. The Bankhead-Jones Act allots funds on the basis of the rural population of each state.

# A STUDY OF THE VAGINAL CONTENT OF PREGNANT BANG-INFECTED COWS FOR THE PRESENCE OF BRUCELLA ABORTUS*

*By* C. P. FITCH, W. L. BOYD *and* LUCILLE M. BISHOP
*University of Minnesota, Saint Paul, Minn.*

The gravid uterus and the udder of Bang-infected cows are the organs most often invaded by *Brucella abortus*. It has been demonstrated, however, by Schroeder and Cotton,[1] Birch and Gilman,[2] and Doyle[3] that this organism may be found in other localizations in the animal body, especially in the lymph-glands. This latter localization does not favor the spread of the disease as it would in organs from which discharges or secretions are eliminated directly outside of the animal body.

The nongravid uterus is especially resistant to infection with *Br. abortus*. Schroeder and Cotton[1] found that when *Br. abortus* was injected into the nonpregnant uterus, it disappeared in a few days. Giltner and Bandeen[5] failed to isolate abortus organisms 24 hours after they had been introduced into the vagina. Repeated bacteriological examinations of non-pregnant uteri from Bang-infected cows are usually negative for abortus organisms, provided the examinations are made 2 to 3 weeks after parturition. There have been scattered reports, however, of finding this organism in the mucosa and submucosa of the uterus several months after parturition. Schroeder,[4] in studies made of the genital tract following abortion or calving, found that *Br. abortus* as a rule was not present after three weeks. Cotton[6] found that the organism may persist in the genital tract for 46 days after calving. Birch and Gilman[2] reported finding it in the genital mucosa or submucosa of four artificially infected cattle, 15, 13, 8, and 7 months after calving, and suggest that further investigation of its persistence in the genital tract of naturally infected cows would appear advisable. Studies in our laboratory have been made of nonpregnant uteri and results have been obtained which would indicate that *Br. abortus* does at times persist in the uterus after it has undergone complete involution (unpublished data).

There is apparently a consensus that the nongravid uterus is only occasionally infected and that the bacteria of Bang's disease

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as a rule do not remain in this organ long after calving or abortion, although they can be found in the vaginal discharges for a short time. Fitch, Delez and Boyd[7] examined the vaginal discharges from 22 infected animals after normal calving or abortion and found that twelve days was the average length of time during which *Br. abortus* was eliminated in the discharges. Twenty-five days after calving was the longest period at which the organism was isolated and five days was the shortest.

Thomson[8] has examined the vaginal estrual mucus of artificially infected heifers for the presence of *Br. abortus*. Eleven infected heifers were examined by him and 176 different specimens, covering 49 periods of estrum, were studied. Only two samples of estrual mucus were found to be positive for *Br. abortus*. Both positive samples were from one animal and Thompson thought that possibly these were due to fecal contamination, as it was customary for this particular animal to struggle while samples were taken, making it difficult to obtain a satisfactory one. This possibility was further supported by the fact that the animal had been infected by feeding cultures of Brucella a few days before the positive samples were taken.

The fact that *Br. abortus* is often found in the gravid uterus of Bang-infected cows suggests the possibility that this organism may be discharged from the uterus prior to calving or aborting. In healthy cows the cervical canal is well sealed from 30 to 40 days after conception and the mucus forming this seal is apparently very effective in keeping bacteria from entering the normal, pregnant uterus. Fitch and Bishop,[9] in a study of 86 pregnant uteri, found the fluids and tissue free of bacteria.

The study reported in this paper is of the vaginal contents of pregnant, Bang-infected cows for the presence of *Br. abortus*. An attempt has been made to determine if *Br. abortus* can be found in the vagina of infected cows at any time during pregnancy. The possibility that pregnant, infected animals may spread the disease by way of the vagina has apparently not been thoroughly investigated. If definite information could be obtained on this point, it would aid materially in methods of control of the disease.

## EXPERIMENTAL

Pregnant, Bang-infected cows were used for this study. Recently infected heifers, as well as old chronic cases of the disease, were examined. Only four of the group were artificially infected, the others being natural infections of this disease. The animals in this study fell into three groups as far as uterine

infection was concerned. Group 1 was a rather large group of animals calving at term which apparently did not have a uterine infection as *Br. abortus* could not be isolated from the placentas. Group 2 carried their calves to full term, although the placentas were infected. Group 3 was made up of animals that aborted before the termination of pregnancy and from which *Br. abortus* was isolated from the placenta and fetus.

Samples were obtained by inserting cotton swabs into the vagina in close proximity to the external *os uteri.* The swabs were then rotated in order to become moistened with mucus, withdrawn, placed in sterile containers and soaked in 20 to 25 cc of physiological saline for several minutes. Five cc of this saline suspension was then inoculated into the peritoneal cavity of each of two guinea pigs. The animals were chloroformed and autopsied four to five weeks later. Cultures were made from the spleen and incubated under 10 per cent carbon dioxide for four days before they were examined for growth. The blood of each pig also was tested for the presence of Brucella agglutinins.

The examinations of vaginal mucus were distributed in some cases over the entire period of pregnancy. The usual routine was to take samples once a month the first, second and third months of pregnancy, twice a month the fourth, fifth and sixth months, once a week during the seventh and eighth months and twice a week during the ninth month of pregnancy. This routine was varied according to the individual animal. If the animal was a heifer with a recent infection of Bang's disease, samples were taken one or two times a week during the fourth to seventh months when abortions were liable to occur. If the study was not made over the entire period, examinations were made close to the calving date. Sometimes the samples were taken at such a late stage of pregnancy that the uterine seal had already broken and parturition had begun.

This study was made on 58 Bang-infected cows, 46 of which gave birth to living, full-term calves and twelve aborted before term. In none of the animals was there a noticeable vaginitis or cervicitis. *Br. abortus* was isolated from the placenta, fetus and colostrum of eleven of the aborting animals. It was isolated from the colostrum but not from the placenta or fetus of one case. The placenta was found to be infected in nine of the full-term pregnancies. The placentas were not obtained in two of the cases, so that the total figure represents nine positive for Brucella out of 44 examinations, or approximately 20 per cent. These figures are comparable to those of Gwatkin,[10] who found 20 per cent infected.

Colostrum was obtained from 44 of the full-term parturitions. Brucella organisms were obtained in 34 (77 per cent).

A total of 440 examinations of swabs taken from the vagina have been made of the 58 reacting animals. Of this group only seven samples were positive for *Br. abortus*. Five of the positive samples were taken within 24 to 48 hours before parturition or abortion and after the uterine seal had broken. One sample taken 21 days before parturition was positive. However, samples taken from this same cow ten days and another 48 hours before parturition were negative. *Br. abortus* was isolated from the placenta of this animal at calving time. Another sample taken 25 days before calving from a second animal was found to be positive. Samples taken 18 days, ten days and 48 hours before calving from this same animal were all negative. The guinea pigs inoculated with the placenta from this animal died within a few days after inoculation. *Br. abortus* was not isolated from the pigs or from direct cultures of the placenta.

Except for the two cases already mentioned, *Br. abortus* was not isolated from any of the swabs taken from the vagina unless the cervical seal was broken.

Twenty of the 58 animals were shown to have had a definite uterine infection. Eleven of the animals aborted and nine calved at term. Negative swabs were obtained from three animals 24 hours before parturition or abortion, from three others 48 hours before, from five 72 hours before, and from three 96 hours before. The uterine seal was not broken in any of these cases at the time of examination. In one of the animals, a swab was obtained five days before parturition or abortion; in two, seven days before; in one, ten days before, and in one, 25 days before. In one case only one sample was obtained. The uterine seal was broken at that time and the sample was positive for Brucella. In some cases swabs were not obtained as close to calving time as could be desired, due to the fact that the animal aborted without showing premonitory symptoms. One hundred and sixty-five vaginal swabs were studied from this particular group of animals.

The evidence that *Brucella abortus* is not found in the vagina prior to calving or aborting until after the uterine seal is broken far outweighs any evidence to the contrary. The two positive samples obtained when the uterine seal was intact out of a total of 440 would indicate that they might have been contaminated with feces or urine. It should be understood that these studies were made on animals not showing noticeable discharges from the vagina. Animals showing such discharges might be exceptions.

## SUMMARY

1. Forty-four examinations were made of the vaginal content of 58 cows reacting to the agglutination test for Bang's disease. *Brucella abortus* was found in two examinations before the seal of pregnancy had broken.

2. *Br. abortus* was isolated from the placentas of 20 of the above animals showing an active uterine infection. Eleven of these animals aborted and nine calved at full term.

3. Forty-six animals calved at term and *Br. abortus* was isolated from the placentas of 20 per cent and from the colostrum of 77 per cent of such animals.

4. These studies indicate that *Br. abortus* is not ordinarily found in the vagina of pregnant, Bang-infected cows until very shortly before an abortion or a normal parturition. This organism, however, appears in the discharges after the seal of pregnancy is broken.

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## DISCUSSION

DR. C. M. HARING: I would like to ask the basis on which Dr. Fitch decided that Brucella infection was present in those animals which he mentioned as giving birth to normal calves at full gestation period.

DR. FITCH: Mr. Chairman, there are two lots in the group which Dr. Haring has mentioned. In one lot, the determination was made by finding the organism in the placenta as a result of cultures and animal inoculations. In the other lot, it was by finding the organism either in the colostrum or by a positive agglutination titre at 1:1,000, or above. There were 75 per cent of the animals that were eliminating the organism in their colostrum.

DR. HARING: It seems to me that the grouping of data on cows of this type might well come under two classes—one in which Brucella is isolated but no lesions in the placenta can be found; the other in which Brucella is isolated, and in which there is evidence of necrosis or other recognized effects of *Brucella abortus* infection.

As stated in my own paper, I believe that the investigation of shedder conditions in cows calving normally and showing no agglutination titre or other evidence of Brucella infection except the isolation of *Br. abortus* from their secretions would be a matter of economic and public health interest.

A year ago, I was invited by Dr. K. F. Meyer, of the Hooper Foundation, to accompany him to New Harmony, Utah, where 21 human cases of melitensis infection had occurred. In one of the herds of Angora goats, consisting of about 1,500 animals, we examined quite a large number of placentas. Out of 75 animals which produced normal offspring during the day when we were present, we found Brucella lesions in over 50 per cent of the placentas. All the kids were apparently full term and vigorous.

DR. FITCH: Did you isolate the organism from the 50 per cent?

DR. HARING: No. The organism was isolated from only two samples. It was impractical to try to isolate the organisms from every placenta, because that material had to be shipped to California, a distance of over 1,000 miles.

DR. FITCH: I have had no experience with goats. We have been looking for Abortus infection in goats in Minnesota for a good many years and have failed to find it. There are not very many goats in the state—I mean four-footed ones. Dr. Boyd and I, a number of years ago, believed that we could determine on gross examination the lesions of Abortus infection in the placenta. We have abandoned that idea. Other people may do it, but we cannot.

DR. R. R. BIRCH: I would like to add my name to the number who can not tell by the examination of the placenta whether *Br. abortus* organisms are present. I can confirm also the statement that in the chronic cases of Bang's disease, in those cows that are calving at full term, there is a considerable percentage of cases in which the Bang bacillus can be demonstrated by the usual guinea pig inoculation method.

DR. HARING: I would like also to add my name to those who are unable to diagnose Bang's disease or Brucella infection by a macroscopic examination of the placenta. Lesions in the placenta accompanying abortions caused by vitamin A deficiency I have observed to be apparently indistinguishable macroscopically from those caused by *Br. abortus*.

DR. B. A. BEACH: Is there anyone here who is familiar with the work that was done in England? I have forgotten just the details of that, but I do know that they reported some two and one-half or three years ago that quite a large number of examinations were made of negative cows in herds that had formerly been positive—that is, herds in which the infection had presumably been eradicated. In fact it had been, as shown by the negative agglutination titres; and in a small percentage, they found the organism in the placenta or uterine discharges at the time of calving. I do not remember the details. I wonder if there is anyone who does remember.

DR. D. H. UDALL: I do not pose as an authority on this subject, but the point you raised is rather interesting to me for the reason that for quite a number of years now—ten, at least—from a herd of about 150 cows, negative to agglutination tests, we make routine examinations of the placentae, gross examinations, and record the findings year after year. Following the period when these cows became negative, guinea pig inoculations were made for I could not tell you how many years, but we never obtained a positive guinea pig from inoculation of placental material from negative cows after this herd became negative.

DR. BEACH: Did you examine the colostrum of those cows?

DR. UDALL: No, just the placentae. In another herd, where similar routine examinations were made, cow after cow was picked up following an exposure, in which there were no gross lesions in the placenta, and a diagnosis was made by guinea pig inoculation.

DR. FITCH: May I add just one final word? Dr. Udall has raised a very important point. We have been working on this in Minnesota, as you know, for a good many years. We get material not only from our experiment animals, but from all over the state. You cannot tell, gentlemen, from the examination of a placenta grossly—as Dr. Haring said, a macroscopic examination—whether or not Brucella infection is present. It may be or it may not be. Guinea pig inoculations are the only method that I know of that would give you that information.

DR. BEACH: Just one more word about the British observations. There was one cow that shed Brucella from the udder 273 days before she reacted. Now, how much longer she actually shed I believe the work did not show, but they picked it up 273 days before she reacted.

We think, at least—and we know it is of some importance—that at the time of an apparently normal calving of many of these old infected cows, most of them are reactors. I do not think they necessarily have to be—that Brucella infection is spread at times quite generally.

DR. HARING: I hate to have the British get all the credit for such work. Drs. F. M. Hayes and E. H. Barger published in *Hilgardia*, vol. 9, November, 1935, observations similar to those mentioned by Dr. Beach.

DR. BIRCH: I recall several years ago that Dr. Carpenter found one cow that shed for a period, I think of about six months, during which she did not show agglutination, but in the examination of a great many cows, we have not found a case of that kind in animals that maintained a curve below 1:50. We have frequently found—and I think that general experience is the basis of many of the reports—that cows which were shedders before the agglutination titre started upward are often offered as examples of cows that do shed despite a negative test. Usually in those cases the test becomes positive in, say, 30 days after calving. I think we want to differentiate sharply between the cow which *maintains a low curve* and still sheds and the one which merely sheds before she builds up an agglutination reaction. We have a great many of the latter. The others, if our experience can be trusted, are exceedingly rare. I would not take the contract of finding one in the next six months.

DR. BEACH: Just one more word on that. In one herd in which we had 13 ceased reactors and about 24 susceptible, they proved to be later susceptible animals. In one of those ceased reactors, after one calving, we picked up the infection in all of the guinea pigs injected, which I think was two to a quarter, and from this one quarter, both guinea pigs were positive and showed the lesions. That cow had been a reactor approximately two years before her titre had gone to negative. She was negative and we could not get a titre over 1:25, yet she did have infection in that quarter.

DR. BIRCH: Was she an artificially infected cow?

DR. BEACH: She was an artificially infected cow that had aborted.

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Government is a contrivance of human wisdom to provide for human wants. Men have a right that these wants should be provided by this wisdom.

—Edmund Burke.

# PARTICIPATION IN PREVENTABLE LIVE STOCK LOSS PROGRAM*

*By* EARLE G. REED, *Omaha, Neb.*

*Supervisor, Agricultural Development, Union Pacific Railroad*
*Chairman, National Live Stock Loss Prevention Board*

The veterinary profession is on the firing line. You who engage in a general live stock practice in agricultural areas are the first line of defense as well as the point of attack, the shock troops and the rear guard in any action, large or small, that has to do with live stock disease, sanitation, health and inspection. Many times you are the sharp-shooters who, unknown and unheralded, snip off and destroy, before it gets started, a minor attack of disease or parasites in live stock that could easily assume epizoötic proportions were it not for the quick, careful and thorough work of practicing veterinarians.

But as great and as far reaching, as effective and as complete as have been the contributions of practicing veterinarians, as well as those employed in large organizations and by the government, in preventing, controlling and stamping out contagious and infectious diseases and parasites, the field of handling preventable losses in live stock production and marketing is still unbounded and uncharted. It presents a real challenge to your profession and through the service that can be rendered a tremendous accomplishment is possible for the live stock industry.

Everyone who has anything to do with live stock production, transportation and marketing knows our efficiency can be greatly improved. Our scientific knowledge, our technic and our practical applications have improved and progressed amazingly in the last few years. This is due largely to excellent veterinary research and exploration. Still we are not reaching the height of our possibilities in successful live stock production and marketing as long as we chalk up each year millions of dollars of loss to farmers and others engaged in the industry.

Figures and statistics are rather odious and they are too easily bent or the light may be turned on particular digits in an effort to prove desired conclusions, so none will be used here. You can "roll your own" figures, but it is sufficient to say that the annual loss in meat production from the time animals are born on the farm to the final number that actually compose the finished meat

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

product of our shops is a staggering sum and if it could all be prevented, it would be sufficient to put every practicing live stock veterinarian in the country on a lucrative annual salary and he would not have to worry about the wolf at the door having a litter of pups. Nor would the veterinarian find himself in the embarrassing position of the doctor who sent a bill to a patient, carefully itemized—so much for medicine, so much for services and so much for a call. The patient returned the bill with a check and a note which read, "I'm enclosing the bill with a check for the medicine and services. I'll return the call." In many, many cases the veterinarian performs a service for which he does not even receive a return call or a come-back invitation.

How can the practicing veterinarian aid in this big problem of preventable losses in the production, handling and marketing of live stock? Being neither oracle nor magician we cannot see things in a crystall ball nor pull white rabbits out of a plug hat but through the perspective of somewhat detached observations we recognize a few facts that have practical applications and may be able to keep the bunny from getting into the hat in the first place. Having taken my collegiate work at an institution that has one of the best veterinary schools in the country, and having done some work under Dr. George H. Glover and Dr. I. E. Newsom, of Colorado State College, as well as having once been a county agricultural agent in a ranching county 65 miles wide and 175 miles long, with no veterinarian within its borders, I can more fully appreciate all sides of this live stock problem.

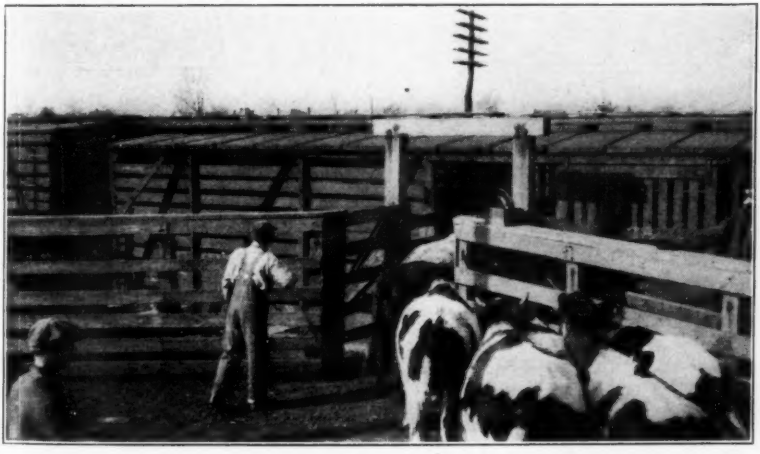

FIG. 1. Loading cattle single file will do much to prevent crowding and bruising.

It is only logical to say that all live stock loss problems begin on the farm—that's where the live stock begins, that's where the breeding, maternity, feeding and problem of care, disease prevention and sanitation begin. The practicing veterinarian can, if he will and chooses to do so, get closer to the average live stock farmer and producer than any other service man or agent unless it might be the banker. But in order to do that the veterinarian has to do two things that too many have failed to do in the past, namely, remove the veil of veterinary mystery from the mind of the farmer and become his friend and adviser, taking away from him through friendly contact and service the too often present idea that the veterinarian is some ghoul waiting to pounce on him and his live stock, order the stock treated, tested, vaccinated, inspected, quarantined or destroyed without cause or reason, just to satisfy the whim of some governmental agency or some unheard of educational cause.

First of all, to benefit in this live stock loss prevention cause, the veterinarian has got to render a service that benefits many. The live stock veterinarian should know more about feeds and feeding of animals than anyone else. Too often he recognizes only the effects of wrong feeding practices and corrects a temporary condition without improving the permanent practices of the farmer. Proper feeding and sanitary precautions and protection in pens, barns and feed-lots are the most important and the veterinarian who advises, suggests and helps a farmer in these

FIG. 2. Canvas slappers should be used in preference to canes or clubs in driving hogs. Poles should be used only for sorting.

things generally gets the call, and the bill paid, when there are professional services to be rendered. Build a strong framework of bone and a healthy body of functioning organs for the animal so it can put on meat and flesh economically and carry it safely to market is a suggestion for every veterinarian to keep out in front and pass on to his farmer friends.

Live stock inspection service with disease and parasite control measures enforced at sale barns and stock yards has provided a new and added field for veterinary service. But too often this becomes, at least in the minds of the farmers and live stock producers, a policing and "crack-down" proposition to be evaded, avoided and tricked if possible, rather than a beneficial and valuable service rendered by a sympathetic, friendly, coöperating animal doctor. That barrier of cold suspicion and indifference between so many farmers and the veterinary profession can be leveled out only by friendly coöperation and helpfulness.

As animals approach maturity and the proper marketing stage, the cost of a loss, mortality or crippling is greater than at any previous time and it is certainly at this period in the process where the practitioner should be most effective in his help to the

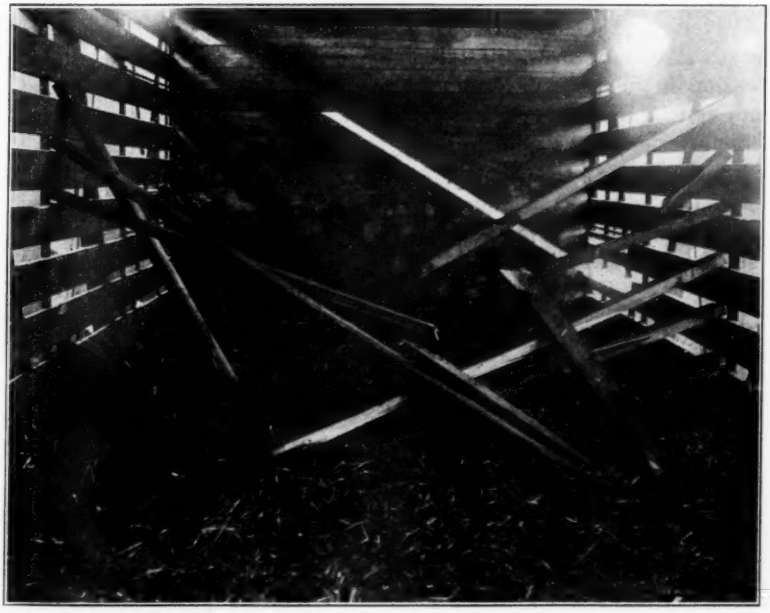

FIG. 3. A poorly constructed, broken-down partition in stock car that may cause much damage to animals.

producer and the industry. Proper feeding and watering before shipping, proper and adequate car or truck bedding—footing, if you please—careful loading and not overloading of either car or truck, partitioning and separating of different classes of animals, what and when to feed in transit and taking the "bunk" out of this question of market fill are all questions that the veterinarian can and should discuss freely, and I mean "free," with his possible and actual clients. If you can help a farmer save a dollar or secure a new dollar he never got before, you may get a part of that dollar, but unless he gets the dollar what chance do you have of getting part of the dollar that some other person already has a full claim upon? That seems to me to be just ordinary main street economics.

FIG. 4. Canvas slapper recommended by the National Live Stock Loss Prevention Board.

Veterinary coöperation and aid in the prevention of live stock losses have real possibilities for building up the veterinary profession in places where its greater appreciation is most needed and can be most beneficial. Direct assistance and aid in the national program of reducing preventable losses in marketing is earnestly solicited. The gospel of farmer-producer self help has to be preached by every veterinarian and all others interested from the cross-roads to the largest packing plant if mutually beneficial results are to be obtained.

Any thing that tends to reduce the producer's loss and increase his income increases the possibility and prospect of your sharing in that income. How much you share will be probably a matter of individual initiative, friendly services, cordial contact—both public and personal relations, in other words. As you serve so may you receive.

On behalf of the National Live Stock Loss Prevention Board and as its chairman, I wish to pay public tribute and give well deserved and meritorious recognition to the fine coöperation and assistance that has been rendered, not only by the American Veterinary Medical Association and its officers, but by hundreds of the practicing members scattered all over the country. We are glad to give you a boost at every opportunity. We know that

on the average you make good on our boast that you and your profession are the first line and the last line in both defense and aggression in the fight to improve live stock conditions. Both education and practice are your weapons. Use both wisely, well and often, for one without the other will fail.

SUMMARY

The veterinarian is the first line of defense as well as the point of attack and the rear guard in problems of live stock diseases, parasites, spread of infections or contagion.

The field of handling and preventing live stock losses from the farm to the market is still wide open and largely uncharted, offering real opportunity for veterinary initiative and practical application of scientific knowledge.

Efficiency in live stock production and marketing through reduction and elimination of preventable losses can be greatly increased. The value in dollars of the annual unnecessary loss reaches a staggering sum.

Live stock loss problems begin on the farm where the live stock begins. Feeding, care, handling, shelter and sanitation are principal questions bearing on later possible losses. The able veterinarian must become a public-relations—even a live stock-relations —man in order to get closer to the producers' problems and serve the industry in a broader way.

Corrective practices, educational propaganda and diplomacy are just as necessary to the veterinarian as good veterinary practices and technic.

New methods of marketing have opened new avenues and ways of serving the producer and the industry. Barriers of suspicion and antagonism must be removed.

The coöperation and assistance of the profession in the campaign of the National Live Stock Loss Prevention Board has been most effective and beneficial.

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## New General Manager for Chappel Bros.

Mr. E. J. Chappel, treasurer of Chappel Bros. Inc., manufacturers of dog foods, has announced the appointment of Walter E. Armstrong as general manager of the company. For the past ten years, Mr. Armstrong has been assistant sales and advertising manager of Chappel Bros. Inc. He was associated with Spratt's Patent Ltd. before he joined the Ken-L-Ration organization at Rockford, Ill.

# HEALTHY CATTLE AS CARRIERS OF COCCIDIA*

*By* HADLEIGH MARSH
*Montana Veterinary Research Laboratory*†
*Bozeman, Montana*

Coccidiosis is the cause of a very considerable loss in young cattle in the northwestern states. While treatment of cattle affected with coccidiosis has been more or less successful, veterinarians have been handicapped in applying preventive measures because the source of the infection and the mode of transmission have remained obscure.

In the northwestern states, coccidiosis is typically a winter disease of young cattle, occurring most frequently in calves. Occasional cases are observed in the spring and summer months, but these constitute a negligible percentage of the total. During and following periods of very severe winter weather, with snow and sub-zero temperatures, many calves develop bloody diarrhea and frequently rectal prolapse. The mortality varies from 10 to 25 per cent. All the clinical cases in any one herd develop within a short period, and the survivors return to their normal condition within a few weeks, but many show retarded development. After such an outbreak, the disease disappears until the next winter, and two or three years may pass before there is a recurrence.

This being, in general, the usual history of outbreaks of coccidiosis in range cattle, the problem of the source of infection and means of carry-over from one season to the next has been difficult to solve. It is known that moisture and a moderate temperature are necessary for the sporulation of oöcysts, and it has been generally considered that the host becomes infected by ingesting feed or water from moist places favorable to the sporulation of the oöcysts. Under the conditions described above, the disease develops in cattle on dry feed and often where the only water available is obtained through a hole in the ice. This being the case, we formerly assumed that the hay might be the source of the infection, by carrying the resistant sporulated oöcysts.[1] It had been observed in Montana that in almost all cases of this disease the affected cattle were being fed irrigated hay, usually alfalfa, or hay grown on naturally sub-irrigated bottom land. The conditions under which the hay was grown would be favor-

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able to the maturation of the oöcysts which might have been deposited on the meadow by cattle fed there during the previous winter. This theory, however, was not considered tenable by Roderick,[2] in view of his observations on the occurrence of coccidiosis in North Dakota, and has since been discarded by us.

The work of Wilson[3] shows that coccidial oöcysts will not resist the drying to which they must be subjected in the curing of hay, which experiments in our laboratory confirm. This conclusion was further checked by attempts to find oöcysts on hay taken from stacks from which cattle were fed at the time coccidiosis developed. Several such samples of hay were examined by the following method: Four hundred grams of the hay was cut into small pieces, covered with 4,000 cc of water, and allowed to stand for one to two days at room temperature. The liquid was then poured off through 20- and 60-mesh screens and centrifuged. The sediment was taken up in sugar solution and again centrifuged. The surface of the sugar solution was examined for oöcysts, with negative results in all cases.

The question then arose as to the possibility that coccidia might be frequent inhabitants of the intestines of healthy cattle, and that clinical coccidiosis develops when the coccidia normally present gain the upper hand due to the intervention of some predisposing factor. This hypothesis seemed to have some justification, based on our observations on sheep. A large number of examinations of sheep feces have been made in our laboratory, the specimens having been taken from sheep from all parts of Montana and under various kinds of management. It has been found that coccidia are almost constantly present in the sheep feces which have been examined, although clinical coccidiosis is rarely recognized in sheep.

The available information in regard to the presence of coccidia in healthy cattle is rather fragmentary. There are several reports in the literature of scattered observations on coccidia in the feces of apparently healthy cattle. Wilson[3] cites Van Nederveen[4] as reporting coccidia in healthy cattle. Davis and Reich[5] report finding coccidia in the colon contents of two out of four slaughtered cattle examined. Becker and Frye[6] report finding coccidia in the feces of three out of 40 calves examined. Wilson[7] found a few oöcysts temporarily in the feces of two out of 19 calves examined preliminary to experimental use. In describing experimental infections in cattle he reports the disappearance of oöcysts from the feces upon recovery from acute coccidiosis. Roderick[2] states that he has been unable to demonstrate coccidia in the feces of healthy cattle.

In our laboratory, examinations of the feces of healthy cattle have produced negative results until recently. In July, 1930, fecal samples were taken from eight dairy calves in the College herd. Examination by the sugar flotation method of Sheather,[8] as used by Benbrook,[9] showed no coccidia. In January, 1933, negative results were obtained by the same method applied to the feces of 32 range Hereford calves on feed at the College.

In February, 1936, an opportunity was offered to obtain information on the persistence of coccidia in the intestinal tract of calves from a herd in which there had been losses from coccidiosis. Sixty range Hereford calves were being fed alfalfa hay in corrals. During a period of extremely cold weather, coccidiosis developed. When the calves were examined on February 19, four had died. Two were passing blood and had rectal prolapse. The remaining animals appeared normal, except for two calves which showed some evidence of diarrhea without hemorrhage, and one thin calf which had recovered from an attack of hemorrhagic diarrhea. Seven calves from this herd were brought to the laboratory for observation. Those selected included the two with rectal prolapse, the two showing slight diarrhea, two that were apparently normal, and the recovered case. One of the two prolapse cases died nine days later, autopsy showing complications of necrobacillosis of the liver and lungs. Three of the remaining six calves were held under observation until September 29, a period of more than seven months, while the other three were held for only two months.

The work done with these calves consisted almost entirely of observations based on fecal examinations. The method of examining feces was a modification of the Lane direct centrifugal flotation method as described by Stoll[10] for counting nematode ova. Instead of 10 gm of feces, the sample used was 30 gm. The 30-gm sample was macerated in 300 cc of water, and the count was made on a 10-cc portion, which represented 1 gm of feces. The flotation medium used was sugar solution instead of salt solution.

The first fecal examinations, made on February 22, showed coccidia present in four of five samples examined. No samples were obtained from the two severe cases. On February 29, fecal samples from all the calves showed the presence of oöcysts. All the calves showed increases in the number of oöcysts in fecal samples during March, with a subsequent drop, until on April 9, the counts were all below 50 per gram, with one animal negative. On April 21, oöcysts were found in all the fecal samples. Three

were below 10 per gram and would have been reported negative if examined by the Benbrook method. At this time the three calves showing the low oöcyst counts were eliminated from the experiment. The three retained for further work included calf 6281, which had a severe case of coccidiosis when received; and calves 6284 and 6287, which had been clinically normal at all times. The two normal calves had much higher coccidial counts than those which had shown symptoms of coccidiosis. Observations on the three calves retained were continued at irregular intervals until September 10, when it was necessary to dispose of them.

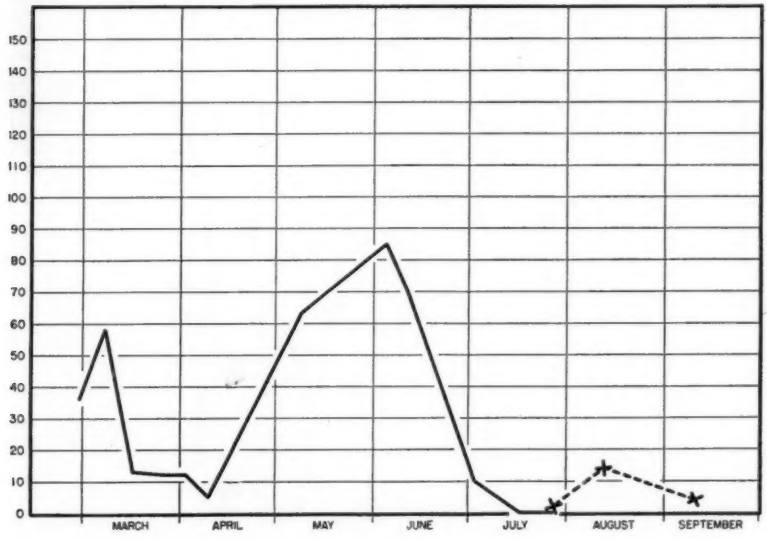


FIG. 1. Graph of total oöcyst counts of calf 6281. Oöcysts per gram of feces. Broken line connects counts made by new method.

Graphs have been prepared showing the oöcyst counts per gram of feces for 15 examinations on the three animals that were carried through the summer. The graph in figure 1 shows the data on calf 6281. From the time when she was received at the laboratory, her oöcyst count was always relatively low, the maximum being much lower than the maximum for either of the normal heifers. Following the low point in April, there was an increase up to a maximum of 85 oöcysts per gram on June 4. The count then fell off to zero on July 16 and July 27. The graphs in figures 2 and 3 show the curves for the two clinically

normal calves (6284 and 6287). The maximum oöcyst count for calf 6284 was 600 per gram on April 2. As in the case of calf 6281, there was a high point in June, with 304 oöcysts on June 4, and 244 on June 11, followed by a drop. The three counts in July were 2, 2, and 5 per gram. The maximum oöcyst count for calf 6287 was 260 per gram on June 11. Again there was a sharp drop down to 2 per gram on July 16 and 27.

When the feces of calf 6281 became negative for oöcysts in July, the question arose as to whether this observation should be interpreted as indicating that the heifer was free from coccidia or whether the method in use failed to show the actual condition.

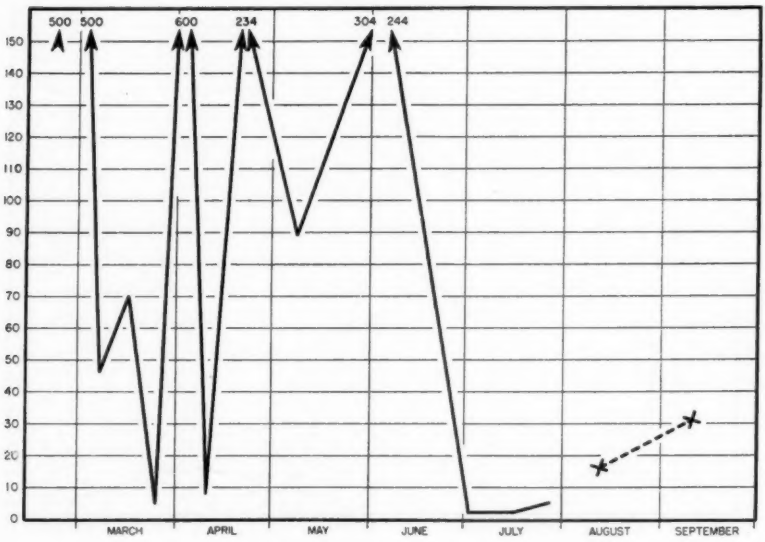


FIG. 2. Graph of total oöcyst counts of calf 6284.

Referring again to the fact that coccidia were regularly demonstrated in the feces of normal sheep, we were reluctant to accept the conclusion that the feces of this heifer were actually free from oöcysts. It is obvious that the amount of fecal material passed by a yearling heifer is many times greater than that passed by a sheep. Therefore, the dilution factor becomes important in interpreting negative findings.

In the Lane method of fecal examination, we were already using a technic about 25 times as efficient as the Benbrook method in detecting coccidia, and were therefore finding oöcysts in samples from recovered cattle which would have been reported nega-

tive if examined by the Benbrook method. We now proceeded to modify our technic in such a way as to multiply by ten the amount of fecal material represented by each microscopic preparation. The amount of material with which we started was increased to 80 gm, which was macerated in enough water to make 400 cc. After standing in the refrigerator for 24 hours, this suspension was poured through 20- and 40-mesh screens. A 50-cc portion was then centrifuged at 1,000 r.p.m. for one minute. The supernatant fluid was poured off and the residue taken up in enough sugar solution to fill a 15-cc centrifuge-tube. This material was then centrifuged and prepared for counting as

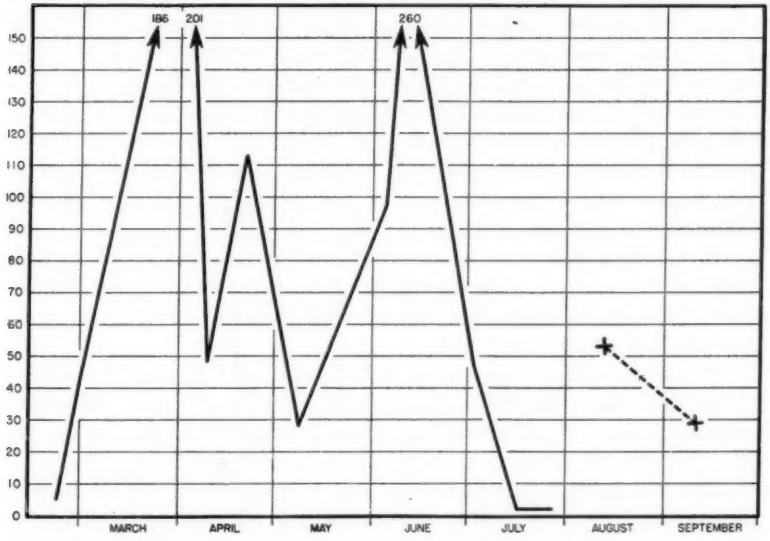


FIG. 3. Graph of total oöcyst counts of calf 6287.

usual. Using this concentration method, the number of oöcysts found in each cover-glass mount was the number in 10 gm of feces. This was the limit of concentration possible, as further concentration made a satisfactory count impossible, due to the amount of suspended material in the final preparation, which interfered with the observation of the oöcysts.

This concentration method was first applied to the fecal sample from calf 6281 taken on June 4, when the old method showed a count of 85 oöcysts per gram. The number of coccidia found by the new method was 728, in a preparation representing 10 gm of feces, or 72.8 per gram. On July 27, when the feces were nega-

tive by the old method, 26 oöcysts were found in 10 gm by the new method, or 2.6 per gram. On August 12 and September 10, the counts by the new method were 14 per gram and 4 per gram.

After September 10, it was necessary to dispose of these three heifers, but it had been shown that coccidia were present in clinically normal heifers as well as recovered coccidiosis cases from the time of the outbreak in February until the approach of the season when clinical coccidiosis might be expected to occur.

In order to obtain further data supporting the hypothesis that the reservoir of infection is in the cattle themselves, fecal examinations were made on young cattle in the College beef herd and on range calves going into their first winter. On June 18, 1936, examinations were made of feces from seven yearling heifers of the College beef herd. By the old Lane method, four of these samples were negative and the other three showed from 1 to 5 oöcysts per gram. The negative samples were then examined by the new concentration method, with the result that coccidia were found in three of the "negative" samples. On September 17, four yearling heifers were examined by the new method, a few coccidia being found in each case.

On October 22, feces from eight beef calves, 10 to 16 months old, were examined. These calves had always been in the College barn. Seven of these samples showed coccidia, ranging in number from 2 to 69 per gram.

On October 29, seven calves, 4 to 8 months old, were examined. They had been in the College barn and in a small adjoining pasture. Six of these calves showed oöcysts in the feces, the number found being from 2 to 29 per gram.

On November 5, samples were taken from 12 out of a bunch of 50 range calves from 4 to 6 months old. These calves had run on dry range since birth. They appeared perfectly healthy in every way. Examined by the new concentration method, coccidia were found in every sample, in small numbers, ranging from one to 34 per gram. These calves were comparable to the calves referred to earlier in this discussion, when 32 feeder calves were examined in January, 1933, with negative results. The difference is that the samples taken in 1933 were examined by the Benbrook method. The twelve range calves examined in 1936 would probably have been negative by the Benbrook method. These calves were held in feed-lots until May, 1936. On May 15, fecal samples were taken from 20 of the 50 animals in this group. Oöcysts were found in 19 of these samples, in numbers much greater than were found in the same calves in November. Conditions for several weeks before the May samples were taken had been favorable

for reinfestation. None of these calves had shown any clinical evidence of coccidiosis at any time.

## Discussion

With the help of a modification of the Lane centrifugal flotation method of examining feces for parasite ova, which enabled us to count the oöcysts in 10 gm of feces in one preparation, we have been able to demonstrate the presence of coccidia in healthy cattle of the age at which coccidiosis usually develops. We have also shown, in three calves from a herd in which coccidiosis had developed, that coccidia were present in the feces in small numbers seven months after the outbreak, although the cattle were in perfect health. These observations appear to justify the conclusion that coccidia may be considered as more or less normal inhabitants of the intestine of cattle, and that clinical coccidiosis develops as a result of some predisposing factor breaking down the resistance of the host. This would provide an explanation for the source of the infection when clinical coccidiosis develops, making it unnecessary to account for the disease by connecting the outbreak with a particular source of infection in the environment of the cattle.

It has been noted by other investigators that coccidia may be found in the feces of healthy cattle, but this has apparently been considered as exceptional. Wilson and Morley[7] suggest that certain immune or tolerant carriers may act as spreaders of the infection. Their conception is that disease results from the exposure of susceptible animals to such tolerant carriers. This could hardly explain the outbreaks of coccidiosis which occur most frequently in the northwestern states, because these outbreaks occur under conditions which are unfavorable to the sporulation of the oöcysts which are discharged by the carriers.

We can present no experimental evidence to support the theory that clinical coccidiosis is the result of lowered resistance to the invasions of a parasite normally present in the intestine of cattle. Field observations, however, indicate that this is the case. This is typically a disease of young cattle, occurring most frequently in calves during their first winter. Calves lack age resistance, if such exists, to the attack of parasites. They are more sensitive than older cattle to changes of feed and environment. During mild, open seasons, when the cattle are grazing more or less throughout the winter, cases of coccidiosis rarely occur. But in severe winter weather, when general resistance is probably lowered by exposure to cold, and when the calves are having their first experience with hay, frequently alfalfa, which

TABLE I—*Oöcyst counts of E. zurnii and E. smithi in six calves.*

| CALF | CLINICAL HISTORY | SPECIES OF COCCIDIA | OOCYSTS PER GRAM ON DATES BELOW | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | 3–7 | 3–16 | 3–25 | 4–2 | 4–9 | 4–21 | 5–8 | 6–4 |
| 6281 | Coccidiosis | *E. zurnii* | 40 | 0 | 0 | 0 | 1 | — | 0 | 0 |
| | | *E. smithi* | 9 | 13 | 12 | 10 | 3 | — | 63 | 85 |
| 6282 | Coccidiosis* | *E. zurnii* | 4 | 1 | 0 | 2 | — | 0 | — | — |
| | | *E. smithi* | 13 | 0 | 150 | 35 | — | 1 | — | — |
| 6283 | Diarrhea | *E. zurnii* | 150 | 10 | 2 | 0 | 0 | 3 | — | — |
| | | *E. smithi* | 6 | 20 | 4 | 2 | 9 | 6 | — | — |
| 6286 | Diarrhea | *E. zurnii* | 2 | 0 | 0 | 1 | 0 | 0 | — | — |
| | | *E. smithi* | 142 | 1 | 10 | 2 | 2 | 2 | — | — |
| 6284 | Normal | *E. zurnii* | 7 | 17 | 0 | 0 | 1 | 2 | 7 | 10 |
| | | *E. smithi* | 38 | 53 | 5 | 600 | 6 | 232 | 82 | 294 |
| 6287 | Normal | *E. zurnii* | — | 6 | 0 | 3 | 7 | 1 | 0 | 0 |
| | | *E. smithi* | — | 112 | 186 | 192 | 37 | 112 | 28 | 96 |

*Recovered.

is very different from the wild grasses, it seems reasonable to assume that the intestinal epithelium may become more susceptible to invasion by the coccidia. As a matter of fact, it is under such conditions that coccidiosis usually develops.

A phase of this work which requires further study is that involving the species of coccidia found in healthy cattle. It has been our experience in examining material from coccidiosis cases that *Eimeria zurnii* is the predominant species. In the work with recovered cases and with cattle which have no history of coccidiosis, *E. smithi* has been the predominant species. *E. smithi* has been present constantly, and in much larger numbers than *E. zurnii*, which has not been found at all in some samples. Oöcysts identified as *E. ellipsoidalis* also were found in small numbers.

Table I shows the counts of oöcysts of *zurnii* and *smithi* in the six calves which were held at the laboratory. Calf 6281 was recovering from an attack at the time the first differential count was made, and showed 40 *zurnii* per gram as compared with 9 *smithi*. Oöcysts of *zurnii* soon practically disappeared from her feces, while those of *smithi* increased in number. Calf 6283 showed some diarrhea when received at the laboratory, and the first differential count on this animal showed 150 *zurnii* per gram as compared with 6 *smithi*. Later counts showed a marked decrease in the number of *zurnii*, while the number of *smithi* remained quite constant. The two clinically normal calves showed at all times a much larger number of oöcysts of *smithi* than of *zurnii*. In 47 differential counts on feces of range and farm calves, *smithi* was found 44 times and *zurnii* 31 times. In only three of these samples was *zurnii* found in larger numbers than *smithi*.

It would therefore appear that possibly the presence of *E. smithi* in the feces of healthy cattle may be of little or no significance as a factor in the development of clinical coccidiosis. However, *E. zurnii* has been found in a sufficient percentage of the samples from healthy animals to justify the conclusion that both species may be considered as normal inhabitants of the intestine of cattle.

## Conclusion

An improved technic has enabled us to demonstrate the presence of coccidia in the feces of healthy cattle. The data presented seem to justify the conclusion that coccidia may be considered as normal inhabitants of the intestine of young cattle in the northwestern states, and that clinical coccidiosis develops

as the result of the effect of environmental factors in lowering the resistance of the host to the parasite.

### ADDENDUM

Since this paper was prepared, it has been found that a report of the presence of coccidia in the feces of healthy cattle has been overlooked. Skidmore, in the *Journal of Parasitology*, xx (1934), p. 126, reported finding coccidia in the feces of cattle, showing no clinical symptoms, in a herd in which coccidiosis had occurred over a period of six years.

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## Serum Producers Launch 1938 Educational Campaign

The 1938 farm educational campaign of the Associated Serum Producers, Inc., designed in the interests of the veterinary profession, will consist of news articles twice a month to newspapers in nine states: South Dakota, Nebraska, Kansas, Minnesota, Iowa, Missouri, Illinois, Indiana and Ohio; illustrated articles on live stock health to 800 selected newspapers in these nine states; one news article per month and eight advertisements each season in 13 farm papers in the nine states named; 18 educational talks in winter and summer, as well as an intensive campaign of spot announcements during the cholera-immunizing season from 14 radio stations, plus special campaigns in coöperation with the state associations in this territory.

## Oldest

The National Woolgrowers' Association, which recently held its 73rd annual convention in Salt Lake City, Utah, is reported to be the oldest national agricultural organization in the United States.

# TRANSMISSION OF THE VIRUS OF EQUINE ENCEPHALOMYELITIS BY AEDES TAENIORHYNCHUS*

*By* LT. COL. R. A. KELSER,† *V. C., U. S. Army*
*Army Medical Research Board, Ancon, C. Z.*

In 1933, the author[1] found that the virus of equine encephalomyelitis could be transmitted from animal to animal by the mosquito *Aedes aegypti*. This constituted the first proof of the ability of mosquitoes to transmit the disease. The results were further interesting in that it was the first time that a filtrable virus, preëminently neurotropic in character, was found to be transmissible by mosquitoes. Since the publication of these findings, the results have been confirmed by a number of investigators and, in addition, several other species of mosquitoes have been proved capable of transmitting the virus.

In June, 1934, Simmons, Reynolds and Cornell[2] announced that they had transmitted the western type virus of equine encephalomyelitis from guinea pig to guinea pig through the agency of *Aedes albopictus*. The details of their experiments were published in 1936.[3]

Merrill, Lacaillade and Ten Broeck,[4] in September, 1934, reported transmission of both the eastern and western virus by *Aedes sollicitans* and the eastern type by *Aedes cantator*.

In May, 1935, Madsen and Knowlton[5] published the results of experiments in which they indicated transmission of the western type virus by *Aedes nigromaculis* and *Aedes dorsalis*. In a footnote in connection with this report it was stated that there was some question regarding the purity of the *A. nigromaculis* employed, it being possible that some *A. sollicitans* were present. However, in a later publication by Madsen, Knowlton and Rowe[6] additional experiments were reported in which apparently definitely identified *A. nigromaculis* and *A. dorsalis* transmitted the disease.

Early in 1935, the author conducted several transmission tests with the western type virus and *Aedes vexans* raised from eggs contained in dried earth collected from areas where this species of mosquito had been breeding. Considerable difficulty was experienced in keeping these mosquitoes alive over the time period ordinarily required in transmission experiments. However, out

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of one relatively large lot of mosquitoes which had fed on three infected guinea pigs, some 35 or 40 survived for eight or nine days subsequent to their infective meals. During this period of survival they were given an opportunity to feed on a total of three normal guinea pigs. One of these three guinea pigs developed encephalomyelitis as a result of of the mosquito bites. It would appear, therefore, that *A. vexans* is capable of transmitting the virus. It was highly desirable to conduct additional transmission tests with this species of mosquito to supplement the one positive instance. However, because the supply of mosquito eggs had been exhausted, it was possible to go on with such tests at that time and before more eggs or larvae could be obtained the writer left the United States for work in Panama. In Panama, *A. vexans* does not occur. Consequently work with this species could not be readily undertaken here.

In July, 1936, the author[7] identified what was apparently the first definitely proved case of equine encephalomyelitis in Panama. The virus in this case was found to be of the so-called eastern type.

With the discovery of the presence of the virus of equine encephalomyelitis in Panama, transmission tests were undertaken with *Aedes taeniorhynchus*. There are 15 known species of *Aedes* in Panama and the Canal Zone, the two most common being *A. taeniorhynchus* and *A. aegypti*. As *A. aegypti* was the species originally used in investigations concerning the mosquito transmission of equine encephalomyelitis, and ample evidence having been accumulated to establish its ability to transmit the virus of the disease, it was decided to conduct experiments with the other common species—*A. taeniorhynchus*.

Through the coöperation of the Army sanitary squad under Mr. J. B. Shropshire, adult *A. taeniorhynchus* and larvae of this species were obtained for the experiments.

It was found that, for some reason, mosquitoes raised from larvae kept under laboratory conditions did not survive nearly so long as adults caught in the field. Because of this, and in view of the fact that adult mosquitoes could be caught in fair numbers in certain localities, most of the experiments covered by this report were carried out with *A. taeniorhynchus* thus obtained.

The transmission experiments were commenced with the eastern type of virus recovered from the case of encephalomyelitis discovered in Panama. The plan of the experiment was, in general, similar to that initially used in the writer's original work[1] with *A. aegypti*.

Adult mosquitoes delivered to the laboratory were placed in feeding cages of the type employed in previous investigations. These cages measure 22x12x12 inches and are constructed of a light wood frame covered with a fine mesh netting (bobinette). The floor of the cage is made of a light-weight insulating board which, together with the wooden framework, is painted white. At one end of the cage the netting is extended about one foot, bunched, and tied by a piece of binding tape. This serves as the door of the cage.

Guinea pigs weighing approximately 250 gm each were used throughout these experiments. Experience has shown that this size pig is best adapted for work with the virus of equine encephalomyelitis.

In attempting to infect the mosquitoes, two guinea pigs were inoculated both intracerebrally and subcutaneously with an emulsion of guinea pig brain tissue containing the encephalomyelitis virus. The subcutaneous inoculation, augmenting the intracerebral injections, was made with the idea that such treatment might make a difference in the time of appearance and persistence of the virus in the blood.

The method utilized for the feeding of the mosquitoes on the infected guinea pigs may be briefly described as follows: Within an hour after inoculation, the two guinea pigs, with their abdomens shaved, are tied out on their backs on an animal-board, a single board being used for the two pigs. The board is then placed in the cage and the mosquitoes given from 20 to 30 minutes to feed. Following this period, the pigs are carefully removed from the cage, precautions being taken to prevent the escape of mosquitoes. It has been our practice to do all of our feeding experiments in a small, well-screened room. Following the feedings, the individual cages containing the mosquitoes are moved out and the room sprayed with an insecticide and closed for about one hour. Except during feeding, the mosquitoes are kept in an insectary, the individual cages, containing supposedly infected mosquitoes, being placed in slightly larger copper-screened cages as an added precaution. A Petri dish containing a fresh 10 per cent glucose solution is routinely placed in each mosquito cage daily.

Feeding of the same lots of mosquitoes on the same guinea pigs is repeated 24 hours after inoculation of the pigs, and thereafter at 24-hour intervals until the pigs are *in extremis* or dead. This will ordinarily vary from three to six days, depending upon the virulence of the virus with which the guinea pigs are inoculated.

In a number of the experiments entering into this investigation, individual lots of mosquitoes, after having fed daily during the course of the disease on one group of two pigs, were subsequently permitted to feed on one or more additional groups of infected guinea pigs before being placed on normal animals. This was done to increase the chance of the maximum number of mosquitoes actually ingesting blood containing virus. As but a portion of each lot of *A. taeniorhynchus* would feed on the same day, it has been evident that more mosquitoes will receive infected blood if the procedure indicated above is followed. However, this cannot always be done with small lots of mosquitoes, as the mortality rate of *A. taeniorhynchus* in captivity is high and if repeated feedings on successive groups of infected guinea pigs are carried too far, there may be few, if any, mosquitoes surviving when it comes time to fed on normal pigs.

The first transmission experiment was commenced on August 25, 1936, starting with a lot of approximately 40 *A. taeniorhynchus* caught on the Fort Amador and Fort Randolph reservations. From August 25 to August 28, both dates inclusive, these mosquitoes were given daily opportunities to feed on two guinea pigs infected with the Panama eastern virus. The pigs were inoculated on August 25 and were dead on August 29.

On August 29, the mosquitoes of this lot were fed on two normal guinea pigs and such feeding continued daily until September 15, 1936, when the last of the mosquitoes died. The result of this experiment was negative, the mosquitoes failing to transmit the disease to the normal guinea pigs.

Between August 28 and September 5, 1936, four additional lots of 35, 46, 52, and 27 mosquitoes, respectively, were used in transmission experiments with Panama eastern virus, with negative results.

On September 7, 1936, a sixth lot of 75 mosquitoes was started in another transmission experiment with the Panama eastern virus. Between September 7 and September 18, 1936, these mosquitoes were given an opportunity to feed on four infected guinea pigs. Commencing September 19, 1936, this lot of mosquitoes was given daily opportunities to feed on a normal guinea pig. On October 1, 1936, this guinea pig showed symptoms of encephalomyelitis and died the following day. Two additional normal guinea pigs were exposed to the bites of this lot of mosquitoes, starting on October 1, 1936. One of these two pigs was dead of encephalomyelitis on October 6 and the second died of the disease on October 8.

Up to this time, the identification of the mosquitoes, as made by the collectors, was accepted by the laboratory and accurate check of individual mosquitoes in a given lot was made only as the mosquitoes died. This was done to avoid increasing the mortality rate which would have resulted from an additional handling of each mosquito when first received. However, a finding in connection with the positive transmission experiment just referred to indicated the necessity of identifying the mosquitoes individually as received, in spite of increasing the mortality rate. A careful check of each mosquito in lot 6, which had proved positive in the transmission experiments, revealed two *A. aegypti,* the others all being *A. taeniorhynchus.* This nullified the particular experiment in so far as transmission by *A. taeniorhynchus* was concerned, because subsequent transmission tests were negative with the *A. taeniorhynchus* in the lot and positive with the two *A. aegypti.* Following this experience, all subsequent tests were carried out with mosquitoes which were individually identified before being used in experiments. This definitely insured that only *A. taeniorhynchus* were used.

As it has been found that certain species of mosquitoes will readily transmit one type of encephalomyelitis and not the other, transmission experiments with the western type of virus and *A. taeniorhynchus* were included in our studies. Within a short time, positive results were obtained with *A. taeniorhynchus* and the western type of virus. A preliminary announcement of this finding was made in February, 1937,[7] and a more detailed account presented before the Medical Society of the Isthmian Canal Zone in April of the same year.

When it became evident that *A. taeniorhynchus* was transmitting the western type of virus, work with the eastern type was temporarily suspended in order that all mosquitoes of the *taeniorhynchus* species received at the laboratory could be utilized in tests to establish clearly the ability of this mosquito to transmit the western type of disease. Thus, between October 5 and December 17, 1936, 14 transmission tests were conducted with *A. taeniorhynchus* and the western type of virus. The number of mosquitoes figuring in each of these tests varied with the supply from daily catches. In a number of instances—as the daily feedings on infected guinea pigs progressed—additional mosquitoes were added to those originally started in a particular lot. This was done to offset the relatively high mortality rate, so that when it eventually came time to feed on a normal guinea pig, there would be in each lot a fair number of mosquitoes which had previously fed on infected guinea pigs.

TABLE I—*Transmission of western type encephalomyelitis virus by Aedes taeniorhynchus.*

| MOSQUITO LOT | MOSQUITOES USED | PERIOD OF FEEDINGS ON INFECTED GUINEA PIGS | ENGORGEMENTS | SURVIVALS FOR TEST ON NORMAL GUINEA PIGS | PERIOD OF FEEDINGS ON NORMAL GUINEA PIGS | ENGORGEMENTS | RESULTS |
|---|---|---|---|---|---|---|---|
| 1 | 30 | 10/7–14 | 21 | 5 | 10/15–27 | 14 | Negative |
| 2 | 68 | 10/12–21 | 97 | 11 | 10/27–11/5 | 27 | G. P. dead 11-20-36* |
| 3 | 61 | 10/13–21 | 37 | 3 | 10/22–26 | 6 | Negative |
| 4 | 65 | 10/14–23 | 59 | 6 | 10/24–11/1 | 31 | G. P. dead 11-2-36* |
| 5 | 115 | 10/16–23 | 94 | 15 | 10/24–11/3 | 46 | G. P. dead 11-4-36† |
| 6 | 130 | 10/17–26 | 122 | 17 | 10/27–11/15 | 35 | G. P. dead 11-16-36† |
| 7 | 112 | 10/21–29 | 106 | 23 | 10/30–11/22 | 89 | G. P. dead 11-23-36† |
| 8 | 153 | 10/26–11/9 | 152 | 15 | 11/10–12/4 | 32 | G. P. dead 12-21-36* |
| 9 | 197 | 10/28–11/9 | 118 | 16 | 11/10–29 | 50 | G. P. dead 12-3-36† |
| 10 | 130 | 11/1–9 | 88 | 9 | 11/10–12/2 | 56 | G. P. dead 12-8-36† |
| 11 | 107 | 11/5–14 | 55 | 11 | 11/15–23 | 7 | G. P. dead 12-9-36† |
| 12 | 89 | 11/6–14 | 46 | 7 | 11/15–12/8 | 18 | Negative |
| 13 | 129 | 11/11–17 | 59 | 20 | 11/18–30 | 45 | G. P. dead 12-16-36† |
| 14 | 127 | 11/15–17 | 33 | 44 | 11/18–27 | 34 | G. P. dead 12-7-36† |

*All mosquitoes of the lot dead when guinea pigs developed symptoms of encephalomyelitis.
†Surviving mosquitoes produced encephalomyelitis in additional guinea pigs.

The total number of mosquitoes entering into each of these 14 transmission tests were respectively as follows: 30, 68, 61, 65, 115, 130, 112, 153, 197, 130, 107, 89, 129 and 127. Table I indicates the results.

As indicated in table I, transmission of the western type of encephalomyelitis from guinea pig to guinea pig by *A. taeniorhynchus* occurred in eleven out of the 14 tests conducted. In the three negative tests there were surviving but 5, 3 and 7 mosquitoes, respectively, when it came time to feed on normal guinea pigs. Apparently it so happened that these particular mosquitoes did not feed on the infected guinea pigs at a time when virus was present in the blood of such pigs. On the other hand, in two of the other tests in which only a few mosquitoes (6 and 9, respectively) survived sufficiently long to test them on normal guinea pigs, the disease was produced.

In three out of the eleven lots of mosquitoes which transmitted the disease (lots 2, 4 and 8), all of the mosquitoes had died before the normal guinea pigs upon which they were feeding developed symptoms of encephalomyelitis. Thus, the mosquitoes of each of these three lots had opportunities to infect respectively but one guinea pig per lot. With mosquito lots 5, 6, 7, 9, 10, 11, 13 and 14, however, some of the mosquitoes in each lot survived sufficiently long to test their ability to transmit the disease to additional guinea pigs. In every instance mosquitoes which had transmitted encephalomyelitis to one guinea pig transmitted it to others upon which they subsequently fed.

The experiments do not indicate the time period elapsing between the ingestion of blood containing virus and ability of the mosquito to transmit the disease. Further tests will be necessary to establish this factor.

It will be noted (table I) that the earliest death occurring in the eleven positive tests was on the ninth day following the exposure of a normal guinea pig to the mosquitoes of lot 4. The mosquitoes of this lot had had daily opportunities to feed on infected guinea pigs over a period of nine days before feedings were started on the normal guinea pigs. At the other extreme it will be seen that the guinea pig exposed to the bites of the mosquitoes of lot 8 did not die of encephalomyelitis until 41 days following the start of exposure to these mosquitoes.

While, in general, a relatively long incubation period occurred with the initial normal guinea pig exposed to the respective lots of mosquitoes, in those positive lots in which there were survivals to test on additional guinea pigs the periods of incubation

were invariably much shorter. It thus appears that multiplication or maturation of the virus in *A. taeniorhynchus* takes place at a relatively slow rate—at least under the conditions obtaining in our experiments. By comparison with the results obtained by the writer[1] in previous experiments with *A. aegypti*, the rate of development of the virus in this species is considerably greater than in *A. taeniorhynchus*. On the other hand, once the multiplication or maturation of the virus in *A. taeniorhynchus* has reached the proper point, the disease is promptly transmitted with fatal results within a few days.

It so happened that in lot 5 a single mosquito survived for ten days after all the others of the lot had succumbed. This lone mosquito was fed once on a normal guinea pig. The pig developed encephalomyelitis and was dead on the fifth day following the bite. This mosquito was then permitted a single engorgement on a second normal guinea pig, resulting in the production of the disease and death of this pig on the sixth day following the bite. With other positive lots it was not uncommon to have several mosquitoes, after finally becoming infectious, bite a normal guinea pig but once and produce encephalomyelitis and death within five to seven days.

All positive transmission results were confirmed by serial passage of the virus through additional guinea pigs, and by histopathologic examination of brain preparations.

## Summary and Conclusions

The mosquito *Aedes taeniorhynchus* is capable of transmitting the western type of encephalomyelitis virus from guinea pig to guinea pig.

Transmission is not mechanical but occurs after multiplication, maturation or, less probably, cyclic change of the virus within the mosquito.

While, under the conditions of these experiments, the precise period of time necessary between the ingestion of virus-containing blood and ability of the mosquito to transmit the disease was not established, it appears that such period is longer in *Aedes taeniorhynchus* than in some other species of *Aedes*, especially *Aedes aegypti*. The evidence, however, indicates that once *A. taeniorhynchus* becomes fully infectious it will readily transmit the disease to guinea pigs with fatal results in a few days. A single mosquito biting a normal guinea pig but once produced encephalomyelitis and death of the pig in five days.

Difficulty in maintaining *A. taeniorhynchus* in captivity over extended periods of time prevented determination of the dura-

tion of infectivity of this species of mosquito. In our laboratory the longest period of time it has been possible to keep *A. taeniorhynchus* alive, subsequent to proving them infectious, is 32 days. Such mosquitoes proved infectious during this period.

Incrimination of *A. taeniorhynchus* brings to eight the total number of species of *Aedes* found capable of transmitting the virus of equine encephalomyelitis.

Several transmission tests conducted in the beginning of this investigation with *A. taeniorhynchus* and the eastern type of encephalomyelitis virus proved negative. However, further tests on this phase of the subject are necessary, and will be carried out, before a definite conclusion is drawn as to the ability or inability of *A. taeniorhynchus* to transmit the eastern type of virus.

ACKNOWLEDGMENTS

As previously indicated, I am greatly indebted to Mr. James B. Shropshire, Field Sanitary Inspector, Panama Canal Department, and his assistants, for most of the mosquitoes used in these experiments. I am also indebted to Mr. Charles L. Pierce, Supervising Sanitary Inspector, and Mr. Carl Brown, District Sanitary Inspector, Health Department, The Panama Canal, for some of the mosquitoes used. Finally, I desire to acknowledge the assistance received from Lt. Col. George R. Callender, M. C., President, Army Medical Research Board, especially in connection with the numerous histopathologic examinations made in the course of this study.

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## Silly

"What's wrong, Henry?" asked his wife.

"My razor," boomed the voice within the bathroom. "It doesn't cut at all."

"Don't be silly. Your beard can't be tougher than the linoleum."

*Pennsylvania Farmer.*

# FOWL LEUKEMIA AND VITAMIN E

## A Preliminary Report*

*By* W. J. BUTLER *and* D. M. WARREN
*Livestock Sanitary Board, Helena, Mont.*
*With the Assistance of August J. Pacini, Minneapolis, Minn.*

Aside from the increasing incidence of fowl leukemia expressed as fowl paralysis, which causes tremendous loss in flocks, particularly throughout the West, but which seems to be increasing and spreading into the East, the problem of fowl leukemia offers a possible introduction to the study of the leukemias that inflict the human and from certain forms of which there is no recovery under any known form of treatment.

Possibly the first published work that pointed to a relation between vitamin-E deficiency and the development of leukemia in the fowl is that of Adamstone.[1] His investigation disclosed that the purposeful destruction of vitamin E in the food of the chick resulted in a condition strikingly similar to leukemia. In addition to a number of symptoms produced in Adamstone's vitamin-E-deficient chicks, which resemble those outwardly manifested by pullets showing leukemic fowl paralysis, a number of the post-mortem findings compare quite closely in both conditions.

Evans and Burr,[2] in their early studies on the anti-sterility vitamin E, demonstrated rather conclusively that the embryos of animals on E-free diets show general under-development, which is most marked in the mesoblast and the tissues derived from it. They conclude that vitamin E is probably needed not only by all fetal tissues but also by the middle germ-layer and its products, which appear to be especially sensitive to vitamin-E withdrawal.

From the mesoblast are derived the bones, cartilages, and connective tissues of the entire body; both the skeletal and the visceral muscles; the entire vascular and lymphatic systems, including the blood-corpuscles, spleen, and serous membranes; the generative organs and the generative elements; and the urinary organs.

Since it has already been shown by Card[3] that hatchability is extinguished in the absence of vitamin E and by Adamstone[4] that there is a high incidence of embryonic chick mortality at the third or fourth day of incubation and again at about the 15th day of incubation; and since it is now amply demonstrated that

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vitamin E is most closely related to reproduction not only in mammals generally but also in fowls, the importance of vitamin E to the development of the embryonic chick and subsequently to the pullet appears evident. In the light of these considerations, it seemed desirable to determine whether vitamin E exerted any curative possibility in fowl leukemia when administered to birds in more or less moribund states. The material used for this purpose was cold-pressed wheat-germ oil,* which was injected hypodermically into the breasts of severely affected pullets. The oil had previously been assayed and was known to contain approximately 40 Pacini-Linn units of vitamin E per gram. At first 2 cc of oil was injected for birds, but after a number of experiments the quantity was reduced to 1 cc. Just previous to the injection, a blood-smear was obtained for a differential leukocyte count.

This study involves about 1,000 paralyzed birds in several western commercial flocks, and will be reported more completely with supporting cytological and other appropriate evidence in suitable journals devoted to poultry science. But the preliminary observations have been so consistently unusual and so exceptionally encouraging as to warrant a preliminary statement not only in the hope of encouraging the enlistment of additional experimental observers elsewhere but also in the trust that perhaps internists in the medical profession may desire to consider the possibility of the use of vitamin E as a therapeutic agent in the prevention and connection of a group of disorders of mesoblastic origin, such as Hodgkin's disease and other leukemias may represent.

The first and most important observation is the quick recovery from paralysis. The paralysis seems to be due mechanically to the encroachment of the nerve from the sheath by an accumulation which resembles a leukocytic infiltration. The infiltration affects the nerves entering the legs but just as frequently may involve the nerves entering the wings. On close inspection, these nerves appear many times enlarged.

Apparently, the injection of wheat-germ oil checks the leukocytic infiltration and may direct its resorption so as to release the fibers of nerves entrenched in its meshes.

It has not been uncommon to observe the restoration of a fairly normal gait to birds that were previously incapacitated so as to preclude any locomotion whatever.

Other changes involve an alteration of the blood-picture which tends to return to normal. The paleness of the wattles, the droop-

*Kindly supplied by Archer-Daniels-Midland Company through the Missoula Mercantile Company, Missoula, Montana.

ing of the wings, and other symptoms are dispelled in many instances. Appetite is restored.

This work has progressed sufficiently to suggest that fowl paralysis or fowl leukemia is a penalty which results from superdomestication of the hen, with its attendant and acknowledged lowering of the vitality of the race.

It is interesting to observe that a somewhat empirical vitamin-E treatment has been suggested for fowl paralysis by Bayon,[5] who treated the condition through forced feeding of the affected birds with lettuce and reported good results. Lettuce can be a fair source of vitamin E. This method of furnishing vitamin E could not be expected to prove as effective as the injection of a definitely standardized source, such as wheat-germ oil.

Two other preliminary observations have occurred with such unerring regularity as to be worthy of special note. First, that the beneficial effects obtained with wheat-germ oil are as effective by the feeding of this source of vitamin E as by its injection. Next, that if, in a flock visited by fowl paralysis, wheat-germ oil feeding is immediately begun, the further incidence of afflicted birds is minimized tremendously; so that there is every indication of preventive effectiveness which offers considerable practical possibilities.

Whereas, Adamstone cautiously avoided suggesting anything more than a strong resemblance to the identity of leukemia with the condition which he produced by feeding vitamin-E-deficient diets, the work for which this announcement is but a preliminary report indicates even more certainly that Adamstone's suggestion is quite likely correct.

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## Married by Proxy

Dr. Carlos J. Cardona (Tex. '36), who is veterinarian in the Department of Agriculture of Venezuela, writes to announce his marriage to Miss Mary D. Rivera, of San Juan, Puerto Rico. The first ceremony took place in San Juan, and was by proxy. The bride then sailed for Caracas, Venezuela, and met her husband there. A church ceremony followed on December 15, 1937.

# A HEMOLYTIC STRAIN OF SALMONELLA ENTERITIDIS FROM A DOG*

*By* LESTER R. BARTO, *Summit, N. J.*

In the past two years, the writer has experienced several similar cases of secondary anemia in dogs, the etiology of which he was unable to determine definitely. The case that is to follow, fortunately for those interested in a scientific terminus, was carried near enough to conclusion to uncover some interesting questions. While this is merely a case report, it is presented for what it may be worth in helping other practitioners to recognize similar cases and finally aid in bringing about a better method of diagnosis and treatment.

The subject was a two-year-old, female elkhound, of high breeding quality. She was in apparently excellent health until one year of age. At this time she was presented to the writer with symptoms of secondary anemia of undetermined cause. The animal was treated successfully with hepatic extract, iron, and an anemia diet over a period of six weeks. The blood-picture returned to normal with a corresponding alertness and activity in the disposition of the anmal.

The dog appeared to be in good health for about one year, or until two years of age, when she was presented with a history and symptoms characteristic of virus distemper (no complications). The temperature was 104.5° F. with a history of anorexia, cough and languidness. Response to repeated administrations of serum was only fair. Ten days after treatment was initiated, congestion of the lungs developed. The temperature was 106° F. Other symptoms included vomiting and cough. Mixed infection serum concentrate and short-wave therapy were given. The temperature remained between 104 and 105° F. Supportive symptomatic treatment, with forced feeding and saline and glucose hypodermoclysis, as a routine measure. Five weeks after illness first developed, two severe nasal hemorrhages 24 hours apart re-

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sulted in visible secondary anemia. The hemoglobin was 40 per cent. A blood transfusion of 600 cc was given. The response was good, the color returned, the edema disappeared, the general circulation improved and the animal took small quantities of food voluntarily. The temperature remained between 105 to 106° F. Hepatic extract 5 cc was given twice daily intramuscularly. Orange juice and Karo syrup, plus an anemia diet force-fed, was retained. Three days after the transfusion, an acute relapse took place, with weakness, vomiting, slight diarrhea, and a temperature of 106° F. On the fourth day after the transfusion, the owner requested euthanasia.

### Autopsy

With the exception of healed scars the lungs are in excellent condition. No visible infection. Heart is pale and flabby. Liver is enlarged, pale and very friable. Spleen is very friable. Kidneys are grayish yellow, with profuse petechial hemorrhages (turkey-egg kidney). The duodenum presents a catarrhal, petechiated inflammation. The jejunum is catarrhal and less swollen, but petechiated profusely. No parasites.

The autopsy was performed immediately after death. Pieces of liver, spleen and kidney were carefully removed and frozen in the ice-cube compartment of an electric refrigerator. The frozen specimens were packed in dry ice and shipped to Lederle Laboratories for culturing.

### Bacteriological Report

The detailed bacteriological report by Dr. Norman J. Pyle is as follows:

From the liver and spleen we were able to isolate pure cultures of *Salmonella enteritidis*. Peculiarly, this strain elaborated a very active hemolysin. In other words, it was hemolytic. This is the first time that we have ever isolated this organism and shown it to have very definite hemolytic properties. The bacteriology of the kidney was negative as far as specific pathogens are concerned.

These results would indicate very clearly that the elkhound died of a *S. enteritidis* septicemia and toxemia. We, of course, cannot eliminate the possibility of there having been present, as a preliminary infection, the filtrable virus of canine distemper. A ferret test on the spleen submitted would have been the only means of determining the presence or absence of this factor. However, because the case in the dog was one of long standing, it is quite possible that the virus content of the spleen, if it were there, had long since been dissipated. Therefore, there was very little practicability in performing such a test.

Recently, we have received a great number of dogs which have proved to have been infected with a hemolytic streptococcic septicemia or toxemia. We are wondering whether in this case, if you had submitted tonsils and lungs, we would have been able to isolate this organism. The fact, however, that the lungs were normal upon autopsy, would have a tendency to eliminate this possibility.

*S. enteritidis* septicemia and toxemia are becoming important disease entities in dogs. We believe that we have not considered them as being of the importance that they deserve. There is no qustion whatsoever but that they do exist as specific entities and are not necessarily preceded by a debilitating canine distemper virus infection.

The history of this case and the results of our bacteriological examination show some very interesting points in common. For instance, this organism was hemolytic. Therefore, we can readily explain why you have had appearances of anemia and a low hemoglobin percentage. Furthermore, it is quite possible that this *S. enteritidis* infection was quiescent or latent and was responsible for the "secondary anemia" affecting the dog approximately one year ago.

The relatively high temperatures of from 104 to 106° F. throughout the course of the disease could be attributed to the toxic effect of the hemolysin. We know very definitely that this organism also elaborates an endotoxin. The immunity against it must be of the nature of an antitoxic antibody as well as one of antibacterial nature. Also, we believe very strongly that infection and immunity with and against this organism are strain specific. You receive temporary response from a transfusion in this case because of the severe toxemia.

The intestinal lesions that you found on autopsy correspond identically to those seen in *S. enteritidis* infection. The autopsy picture as a whole was quite typically that of a severe toxemia. By that, we mean an apparently albuminous degeneration of the spleen and the liver; also, a pronounced nephritis.

Again, we do not feel that we can definitely eliminate the possibility of a coexistent hemolytic streptococcic infection. Your autopsy showed lung scars. Is it not possible that the animal had completely recovered from lung lesions due to hemolytic streptococci? To repeat, had we had the lungs and tonsils for examination, we might have found that organism therein.

Just a word about the use of canine distemper serum in this case. We have intimated that the infection and immunity are strain specific. Therefore, the *S. enteritidis* antibody in the canine distemper serum used by you would have been of only mild alleviating importance.

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# ACETONEMIA*

*By* JAMES K. STROCKBINE, *New Cumberland, Pa.*

The apparently beneficial influence of estrum on a case of acetonemia prompts me to inquire if this is a common experience and also if therapeutic measures might not include massage of the ovaries and uterus, expression of corpora lutea and administration of some glandular product, such as ovarian extract.

The subject was a five-year-old Guernsey cow in very good condition that freshened about June 25, 1937. The cow was apparently normal the first ten days following calving, but from then on she exhibited an unsteady gait, impaired appetite, decreased milk-flow and a strange but frequent bawl. The severity of these symptoms varied during the day and from day to day, but at no time did they entirely disappear or become so severe as to cause the cow to go down. These symptoms were continuous,

*Received for publication, December 31, 1937.

with moderation following treatment until she came in heat, about September 1, when there was an immediate return to normal.

On July 16 and 19 and August 9, the urine was strongly positive to the acetonemia test. On July 16, 500 cc of dextro-calci-nate solution (Jen-Sal) was administered intravenously with quite marked improvement that lasted about 24 hours. On July 19, inflation of the udder was tried along with the intravenous administration of 250 cc of a 50 per cent dextrose solution. The results were about the same as with the dextro-calci-nate solution, with the exception that the beneficial results lasted longer. Inflation of the udder was repeated on August 9, and chloral hydrate was left for the owner to administer by mouth. The results here were transient as before. On August 15, barium chloride, tartar emetic and strychnine tablets were prescribed with no improvement.

## MARE ADOPTS CALF*

*By* W. J. BUTLER, *Helena, Mont.*
*State Veterinary Surgeon of Montana*

The accompanying photograph, showing a mare nursing a calf, was taken by Mr. A. M. Moore, vice-president of the Montana Livestock Sanitary Board, at his ranch at Marion, Mont. In all

FIG. 1. Mare nursing calf.

of my experience, I never before saw a mare adopt a calf. The history is a simple one. The mare lost her colt. The calf is the ranch milk cow's calf and was adopted by the mare. The calf apparently is doing exceptionally well on mare's milk.

*Received for publication, January 24, 1938.

# FIBROSARCOMA FRACTURE SEQUEL INVOLVING PREMAXILLA OF HORSE*

*By* J. F. WITTER, *Orono, Maine*
*Animal Pathologist, University of Maine*

A comminuted fracture of the premaxilla of a horse while working in the Maine woods, early in the spring of 1937, developed into a tumor involving the upper incisor teeth and protruded beyond the upper lip. The case was examined by Dr. S. W. Stiles, of Falmouth Foreside, Maine, in April. By June, the tumor had doubled in size and Dr. Stiles was asked to remove it.

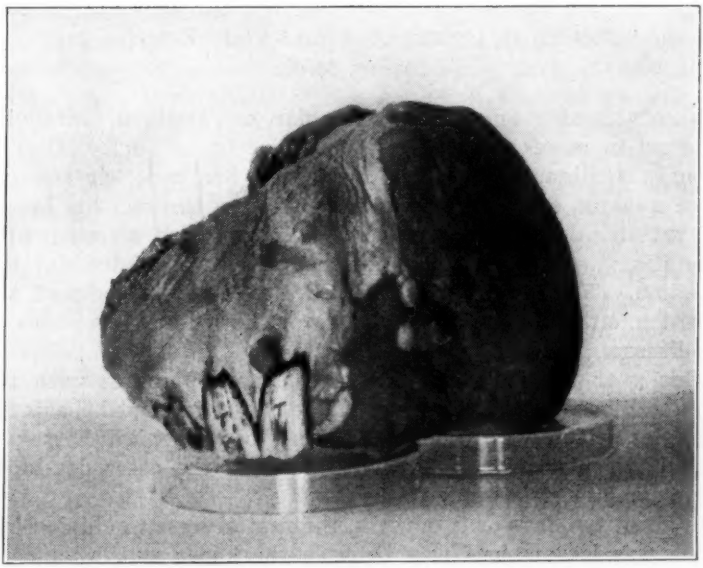

FIG. 1. Side view of tumor showing extent of mucous membrane.

The horse was given 2 ounces of a 25 per cent solution of chloral hydrate intravenously and the tumor-fracture was removed, including the upper incisor teeth (see figure 1) and most of the premaxilla. The inner surface of the upper lip was sutured to the hard palate, the wound washed daily with hydrogen peroxide, and the stitches allowed to slough. The horse was eating within four hours, was put to work by July, and by October had gained 100 pounds to equal its original weight.

*Received for publication, January 20, 1938.

This growth weighed about 4½ pounds. The premaxilla was apparently split at the symphysis, causing one incisor tooth to be lost, and the remainder to be widely separated by the tumor. About one-half the surface was covered with normal mucous membrane and the remainder was the exposed tumor. Histological examination showed the tissue to be a mixed tumor, of a mildly malignant type, being largely a fibrosarcoma with some small areas suggesting an adenoma.

## IRREGULAR STRANGLES TRANSMITTED BY COITION*

*By* CHARLES H. HAASJES, *Shelby, Mich.,*
*and* DONALD H. DICKIE, *Assistant State Veterinarian, Lansing, Mich.*

A considerable amount of irregular or "bastard" strangles occurred in mares in the territory adjacent to Shelby, Oceana County, Michigan. A stallion-owner who has two Belgians and one Percheron evidently spread the infection through his breeding activities. The two Belgian stallions were affected with strangles during the spring months and were treated for the infection. The evidence is that all three stallions spread the infection, although the Percheron never showed any symptoms of the disease.

This year the stallion-owner bred over 200 mares with the three stallions. About a dozen of these mares developed "bastard" strangles which was thought at first to be genital horse-pox (coital vesicular exanthema) until some of the cases developed grave symptoms.

In about ten days or two weeks, the first abscess would develop, which was located half way between the upper and lower commissures of the vulva and about one inch from the edge of the lip. After the appearance of the first abscess, more would develop around the anal and perineal regions and on the udder. For one mare, nine calls were made to lance 13 abscesses. Usually these abscesses were large and would contain as much as two quarts of pus. The whole hand could be introduced into the abscess cavity after the pus had been drained out. Examination of the stallions did not disclose the reason for the irregular form of strangles and the bacteriological examination made by the Michigan Depart-

*Received for publication, September 2, 1937.

ment of Agriculture laboratory disclosed a streptococcus which could not be differentiated from *Streptococcus equi* found in regular strangles. In the suckling colts the infection produced the regular form of strangles.

The irregular form of strangles has been rather infrequent during recent years. Therefore, we desire to report this outbreak in Michigan.

## Four Ohio State University Deans Honored

Ohio State University honored the deans of four departments with a dinner and reception at the Faculty Club, on January 21. Each of the four deans is this year president of his national professional association. Dr. Oscar V. Brumley, dean of the College of Veterinary Medicine, is president of the American Veterinary Medical Association. Dr. John H. J. Upham, dean of the College of Medicine, is president of the American Medical Association. Dr. Harry Semans, dean of the College of Dentistry, is president of the American Association of Dental Schools. Dr. Herschel W. Arant, dean of the College of Law, is president of the Association of American Law Schools.

## United States Civil Service Examination

The United States Civil Service Commission has announced an open competitive examination for the position of Junior Scientific Aid (Parasitology) in the Bureau of Animal Industry, Department of Agriculture, at a salary of $1,440 per year.

The necessary application forms may be obtained from the Secretary, Board of U. S. Civil Service Examiners, at any first-class post office, or from the U. S. Civil Service Commission, Washington, D. C. Applications must be on file not later than February 28, 1938 (east of Rocky Mountains), or March 3, 1938, for western points.

## East Likes Lamb

According to the Institute of American Meat Packers, three-fourths of the lamb in the United States is produced west of the Mississippi River and nearly one-half of all the lamb eaten in the United States is consumed in New York, New Jersey and Pennsylvania.

SPOTTED FEVER III. The identification of *Dermacentroxenus rickettsi* and its differentiation from the non-pathogenic rickettsiae in ticks. Henry Pinkerton and George M. Hass. Jour. Exp. Med., lxvi (1937), p. 729.

Comparative studies were made of the microörganisms present in *Dermacentroxenus variabilis* ticks, some of which served as a control series, while the remainder were exposed to infection with *D. rickettsi* and thereafter maintained under various conditions. All female ticks contained in their ovaries coccoid intracellular microörganisms. About 50 per cent of all ticks, after feeding, contained rickettsia-like microörganisms in variable numbers in nearly all organs. The group of ticks exposed to infection with the virus of spotted fever, in addition to the above-mentioned microöganism, usually harbored large numbers of *D. rickettsi*, distinguishable with certainty from the non-pathogenic organisms only by their localization in nuclei of tick cells. No influence upon the size, number or distribution of either the non-pathogenic rickettsiae or *D. rickettsi* in ticks was attributable to refeeding, variations of the temperature of incubation, or variations of the length of the period of incubation. It is concluded that the non-pathogenic rickettsiae which occur in *D. variabilis* ticks have no well-defined relationship to *D. rickettsi*, since they differ from the latter organism not only in absence of virulence and immunizing properties but also in their distribution in tick tissues and inability to multiply in the nuclei of cells.

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TOLERANCE OF ANIMALS TO PYRETHRUM. E. R. DeOng. Jour. Econ. Entomol., xxx (1937), p. 921.

The experiments show that great excesses of spray (pyrethrum in oil) were harmless when fed to feeding lambs and pregnant ewes and to lambs born to same. Sprayed forage showed no repelling action; sheep in many instances preferred the sprayed forage. Any spraying done on the range with this type of spray is therefore considered harmless to sheep. When pyrethrum is

exposed to sunshine and open air for five hours, only 32 per cent of the normal activity of the active principle detrimental to insects remains. Ten minutes of exposure to a sun-lamp resulted in a loss of 49 per cent, while exposure in a darkened room for seven hours resulted in the normal activity remaining at 95 per cent.

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COMPARISON OF THE WEIGHTS OF SUPRARENALS OF DOGS IN ESTRUS, PREGNANCY AND LACTATION. Dan. D. Baker. Jour. Morphol., lxii (1937), p. 3.

A comparative study was made of the weights of the suprarenal glands of 188 dogs in and near estrus, 76 pregnant, 23 pseudo-pregnant and 12 lactating dogs. There was a significant hypertrophy of the glands of estrous animals in only a few weight groups. As the animals passed into metestrus, the suprarenals rapidly decreased in size. There was a slight hypertrophy of the suprarenals of the pregnant dog when the net body weight was considered, but there was practically no hypertrophy of the glands when the total body weight was used in comparison. There was only slight hypertrophy of the suprarenals during pseudo-pregnancy and no hypertrophy in lactating dogs. The suprarenals were slightly smaller in metestrus than in diestrus.

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THE IMMUNITY TO THE HOOKWORM, ANCYLOSTOMA CANINUM, ACQUIRED BY DOGS AS A RESULT OF PREVIOUS INFESTATION. G. F. Otto, K. B. Kerr and J. W. Landsberg. Abst. Jour. Parasitol., xxiii (1937), p. 560.

Young mongrel dogs were protected against overwhelming infections with hookworm (*Ancylostoma caninum*) by 25 to 30 immunizing infections, gradually increasing from 15 to 120,000 larvae over a period of four to five months. About three weeks after the last immunizing infection, these animals and previously uninfected litter-mates were given test infections of 120,000 to 200,000 larvae. None of the four immunized animals showed any appreciable effects from the test infections. Two immunized animals in one litter, given test infections of 120,000, harbored only 118 to 203 hookworms when killed three weeks later, whereas the previously uninfected litter-mate died on the ninth day and 13,400 hookworms were recovered from its intestine. Two immunized and unimmunized animals in another litter gave similar results. The immunity to hookworm infection seems to be essentially the same as that which has been demonstrated in

other worm infestations. Slowly increasing doses over a long period often must be given on account of the debilitating effect of this blood-sucking parasite.

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STUDIES ON THE COURSE OF TRICHOSTRONGYLE INFESTATION IN SHEEP. H. S. Cameron and M. A. Stewart. Abst. Jour. Parasitol., xxiii (1937), p. 569.

Lambs weaned from ewes at 24 hours of age were reared on bovine milk and maintained under quarantine conditions in quarters with concrete floors that were swept daily and disinfected weekly. They were free from trichostrongyles as determined by fecal examination. At various ages, ranging from one to four weeks, they were infested orally with a known number of infective larvae in gelatin capsules. Non-infested lambs mingled with the infested ones as a control on reinfestation. The larvae consisted of *Ostertagia circumcincta* and *Trichostronglylus colubriformis*. A few *Oesophagostomun sp.* were present. Fecal samples were taken daily. Results indicated that lambs one week old were susceptible to trichostronglyes and that the intrinsic incubation period of the parasite, including the passage of the eggs along the alimentary tract, was approximately 20 days. Daily fluctuation in egg-counts was great. In all instances negative phases occurred, followed by sudden rises and then equally sudden drops. The shortest duration encountered was 120 days, followed by 77 days of negative examinations.

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STUDIES ON THE SEROLOGICAL RELATIONSHIP OF THE RABBIT VIRUSES, MYXOMATOSIS AND FIBROMA. J. C. G. Ledingham. Brit. Jour. Exp. Path., xviii (1937), p. 436.

Rabbits infected with fibroma virus develop agglutinins in their sera which act both on myxoma and on fibroma. On the localizing action *in vivo* of such myxoma agglutinins probably depends the resistance exhibited by fibroma-recovered animals to superimposed infection with myxoma. Rabbits which recover, as they may do, if rarely, from a myxoma infection naturally contracted, and rabbits infected with the neuromyxoma variant of Hurst contain in the sera agglutinins both for myxoma and fibroma. The fibroma and myxoma viruses are probably not identical when serologically analyzed, the former being distinguished by its lack of antigen fraction capable of stimulating the formation of myxoma-neutralizing antibodies.

SOME OBSERVATIONS ON THE EPIDEMIOLOGY OF THE DOG HEART WORM DIROFILARIA IMMITIS. Harold W. Brown. Abst. Jour. Parasitol., xxiii (1937), p. 570.

Examinations of dogs for *Dirofilaria immitis* were made in rural and urban areas in and about Savannah, Georgia. The examinations disclosed that this parasite was present in as many as 80 per cent of the dogs in some areas. Studies made on the type of dogs most commonly infected indicated that the usual text-book statement of a higher infection rate among short-haired dogs than among long-haired dogs does not hold for the region studied. Observations were made on the development of the microfilaria in dog fleas.

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FURTHER STUDIES OF THE AGENT OF THE ROUS FOWL SARCOMA. A. Centrifugation experiments. B. Experiments with the lipoid fraction. E. M. Fraenkel and C. A. Mawson. Brit. Jour. Exp. Path., xviii (1937), p. 454.

Concentration of the active agent of the Rous fowl sarcoma is not invariably obtained in the deposit after centrifugation for two-hour periods at 15,000 rpm. There is no relation between the tumor-producing activity of a preparation and the number of "elementary bodies" visible therein. Small variation in the quantity of Rous fowl sarcoma extract injected produces tumors of variable size and time of appearance in the same bird. No agent could be obtained by means of extraction with acetone and carbon tetrachloride. These extracts seemed to contain an inhibitor, whereas the agent may be obtained from the residue remaining after extraction of the dried Rous fowl sarcoma tissue.

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THE CULTIVATION OF RABIES VIRUS IN TISSUE CULTURE. H. Bernkopf and I. J. Kligler. Brit. Jour. Exp. Path., xviii (1937), p. 481.

Experiments are reported in which it was possible to cultivate fixed rabies virus in media containing rat or mouse brain embryo. Serial subcultures were obtained in a medium consisting of chicken plasma covered with Tyrode-serum (rabbit serum may be used, although human serum is better). The virus cultures incubated at 30° C. or 37° C. increased in virulence up to about the sixth or seventh day. Thereafter the virulence declined and disappeared by the 20th day. Formalin (1:1,000) inactivated the cultures if kept one hour at 37° C. and then for

## Regular Army

Captain William E. Jennings is relieved from further assignment and duty as student, Army Veterinary School, Army Medical Center, Washington, D. C., effective on or about January 1, 1938, is then assigned to station at Front Royal Q. M. Depot, Front Royal, Va., and will proceed at the proper time to Lexington, Ky., for temporary duty for a period of approximately five months, for the purpose of pursuing a course of instruction in breeding and clinics under Dr. Dimock, at the University of Kentucky, under the provisions of section 127a, National Defense Act, as amended. Upon completion of this temporary duty, Captain Jennings will proceed to Front Royal, Va., for duty. He will report to the officer in charge of the Lexington Remount Area, for additional duty.

Captain Harvie R. Ellis is assigned to duty at Spokane, Wash., in addition to his other duties atFort George Wright, Wash., for the purpose of doing veterinary inspection of supplies of animal origin originating in Spokane, and will report by letter to the commanding general, port of embarkation, San Francisco, Calif., for necessary instructions.

Lt. Colonel Chauncey E. Cook is detailed to duty as attending veterinarian, Fort Washington, Md., in addition to his other duties in Washington, D. C.

The promotion of the following named officers to rank of captain, from dates opposite their names is announced:

1st Lt. William F. Collins, Dec. 1, 1937.
1st Lt. Ray S. Hunsberger, Dec. 1, 1937.
1st Lt. Daniel S. Stevenson, Dec. 1, 1937.
1st Lt. Velmer W. McGinnis, Dec. 17, 1937.

## Veterinary Corps Reserve

NEW ACCEPTANCES

Cornwell, James Ira.....1st Lt...435 Biltmore Ave., Asheville, N. C.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Bruner, Alta R.....1st Lt...Ft. Benj. Harrison, Ind. CCC Dist.
Omalden, Rudolph O.....1st Lt...Charleston Dist., Charleston, W. Va.
Sheetz, Harold O.....1st Lt...Fort Knox Dist., Fort Knox, Ky.
Hasson, David S.....1st Lt...Chicago Q. M. Depot.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

Willis, Robert Leon.....1st Lt...Fort Oglethorpe, Ga.
Tuttle, Marion F.....1st Lt...Fort Benj. Harrison, Ind.
Schwiesow, Carlton W.....1st Lt...Camp Custer, Mich.

## SOUTHERN STATES VETERINARY MEDICAL ASSOCIATION

An interesting and instructive meeting of the Southeastern States Veterinary Medical Association was held at the Ansley Hotel, Atlanta, Georgia, October 14-15-16, 1937. This meeting is one that is looked forward to each year by the veterinarians of the southeastern states and their wives, as an occasion to meet friends professionally as well as socially. Officers in charge were Dr. M. M. Leonard, of Asheville, N. Car., President, and Dr. M. R. Blackstock, of Spartanburg, S. Car., Secretary-Treasurer.

Papers of special interest were presented by Dr. C. E. Bild, of Hallandale, Fla., on "Heart Worm Infestation"; Dr. Mark Welsh, of College Park, Md., on "Encephalomyelitis" and "The Maryland Plan of Hog Cholera Control"; Dr. H. J. Shore, of Fort Dodge, Iowa, on "Skin Diseases of Small Animals"; Dr. B. C. McLean, of Aiken, S. Car., on "Equine Strongylosis," and Dr. W. B. Hirleman, of Waynesboro, Ga., on "Swamp Fever in Mules."

The medical profession was represented by Dr. Chas. H. Leach, of the Rockefeller Foundation staff, Montgomery, Ala., and Dr. T. F. Sellers, of the Georgia State Board of Health Laboratory, who gave reports on rabies and rabies research. Dr. John Williams, also a physician, also gave an interesting talk on "The Veterinarian's Place in Public Health Programs."

Dr. Oscar V. Brumley, president of the American Veterinary Medical Association, was present and gave a talk at the banquet held the evening of the first day. The clinic was held on the morning of the third day, at the hospital of Dr. L. J. Hopping. It was of unusual interest and was well attended.

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Adory, of Auburn, Ala.; secretary, Dr. L. A. Mosher, of Atlanta, Ga., and treasurer, Dr. M. R. Blackstock, of Spartanburg, S. Car.

The 1938 meeting will be held in Knoxville, Tennessee.

L. A. MOSHER, *Secretary.*

## NATIONAL ASSOCIATION OF BUREAU OF ANIMAL INDUSTRY VETERINARIANS

The twentieth annual meeting of the National Association of Bureau of Animal Industry Veterinarians was held at the La Salle Hotel, Chicago, December 1-2, 1937. Dr. W. A. Sullivan, of Cheyenne, Wyo., presided.

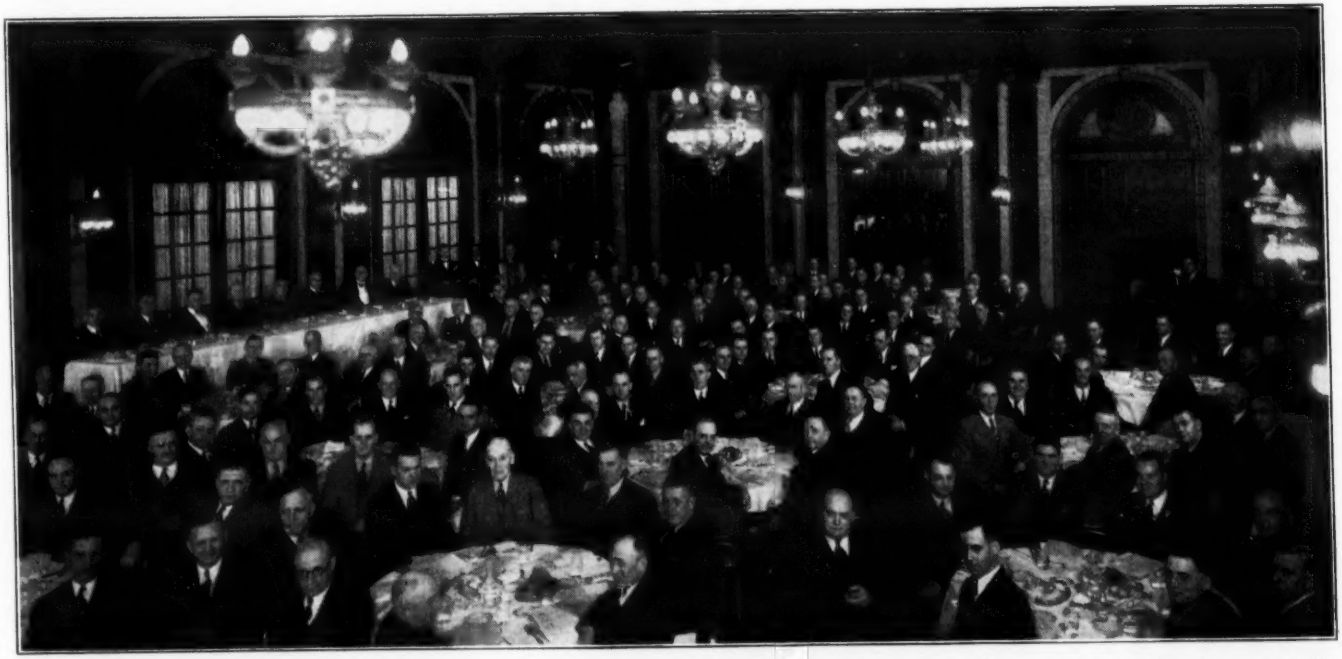
After transacting considerable business, officers for the ensuing year were elected as follows: President, Dr. R. W. Culbert, of Saint Louis, Mo., and vice-president-at-large, Dr. A. J. DeFosset, of Columbus, Ohio. The Executive Board reappointed Dr. F. A. Imler, of Kansas City, Kan., as secretary-treasurer.

The annual banquet was held in the Red Room of the La Salle Hotel the evening of December 2. There were 157 in attendance, including Bureau veterinarians and their friends. Dr. J. S. Healy, of Madison, Wis., presided as toastmaster in his usual efficient manner. The guest speakers included Dr. R. W. Smith, State Veterinarian of New Hampshire, and president of the United States Live Stock Sanitary Association; Dr. Oscar V. Brumley, president of the American Veterinary Medical Association; Dr. A. W. Miller, Assistant Chief of the U. S. Bureau of Animal Industry, and Mr. Douglas Malloch, the "poet who makes living a joy."

## VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The annual meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, on Wednesday evening, December 1, 1937. In the absence of President MacKellar, Dr. L. W. Goodman, Vice-President, occupied the chair. The meeting was turned over to Dr. E. R. Blamey, chairman of the Program Committee, who had arranged for a symposium on distemper.

The first speaker was Dr. N. J. Pyle, who gave a very instructive talk on the laboratory work with distemper and the progress



BANQUET, NATIONAL ASSOCIATION OF BUREAU OF ANIMAL INDUSTRY VETERINARIANS, LA SALLE HOTEL, CHICAGO, DECEMBER 2, 1937.

being made in the standardization of the virus. Dr. George Watson Little and Dr. James S. Crawford spoke on the prophylaxis of distemper, giving many practical hints and suggestions in connection with the use of the biologics that are now available to the veterinarian for the purpose of immunizing against distemper.

Dr. Joseph A. Millar and Dr. C. P. Zepp covered the field of diagnosis in a very able and comprehensive manner. Biological treatment in its various phases was presented by Dr. O. E. McKim, who discussed the use of whole blood from dogs that were carrying natural antibodies, as well as commercially prepared serum, either natural or hyperimmune. He also stressed the importance of the use of liver extract, both in its action in raising the hemoglobin content as well as the leokocyte index. He declared that regardless of the method of administration, whether it be intravenous, intramuscular or subcutaneous, the results are always obtained.

Dr. J. Elliot Crawford then gave the medical treatment in his own genial manner, which was very interesting. The discussion was completed by Dr. M. L. Morris, who summed up the more important points made by the previous speakers and added many helpful suggestions with special reference to the hematology of distemper. This concluded a very thorough discussion of this common and much dreaded disease, so called distemper.

The following officers were elected for the ensuing year: President, Dr. Robert S. MacKellar, Jr.; vice-president, Dr. L. W. Goodman, and secretary-treasurer, Dr. Joseph B. Engle.

JOSEPH B. ENGLE, *Secretary.*

## MAINE STATE VETERINARY ASSOCIATION

The quarterly meeting of the Maine State Veterinary Association was held at Hallowell, on January 5, 1938, with 21 veterinarians in attendance. Dr. R. E. Libby, of Richmond, presided.

During the business session, Dr. C. F. Davis, of Rumford, president of the New England Veterinary Medical Association, proposed some plans for the annual meeting of that organization, to be held next fall in New Hampshire.

Dr. C. L. Blakely, of the Angell Memorial Hospital, Boston, Mass., spoke on "Technic in Small-Animal Examination." Dr. R. E. Whitcomb, of Belfast, gave a paper on "The Physiology of the Liver." Dr. C. M. Merrill, of South Paris, presented a paper on "Newer Methods in the Treatment of Fractures."

A resolution was proposed by Dr. P. R. Baird, of Waterville, and adopted by the Association, urging the State Division of Animal Industry to require all live stock trucks and common carriers to be disinfected at regular intervals.

The annual election put into office: President, Dr. J. F. Witter, of Orono; vice-president, Dr. S. W. Stiles, of Falmouth Foreside; secretary-treasurer, Dr. A. E. Coombs, of Skowhegan; Executive Committee: Dr. C. L. Ryan, of Dexter; Dr. E. E. Russell, of Farmington, and Dr. C. F. Davis, of Rumford.

J. F. WITTER, *A. V. M. A. Resident Secretary.*

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## UNIVERSITY OF PENNSYLVANIA CONFERENCE OF VETERINARIANS

The annual Conference of Veterinarians was held at the School of Veterinary Medicine, University of Pennsylvania, Philadelphia, January 4-5, 1938. It marked the 38th consecutive meeting for this purpose, inaugurated in 1900, by Dr. Leonard Pearson, then dean of the veterinary faculty. The program presented at this meeting was planned with the idea of following the concepts and purposes of Dean Pearson in presenting, before the practicing veterinarians of this commonwealth and nearby states, some of the recent developments in technical and research phases of the subjects, as well as clinical reports and matters of general interest. The attendance was estimated at somewhat over 100.

The Conference commenced with clinics in the forenoon of the first day. The afternoon session was distinguished by papers from Drs. M. F. Barnes, Director of the Pennsylvania B. A. I. Laboratories, "Essentials Necessary in the Practical Control of Bang's Disease"; Joseph Klauder, "Bacillus of Swine Erysipelas Infection in Man," and Donald G. Lee, "A Practical Discussion of Blackhead or Enterohepatitis of Turkeys." At the evening session of the same day we were privileged to hear Prof. S. S. Huebner, of the Wharton School, University of Pennsylvania, and president of the American College of Life Underwriters, on the topic, "The Human Life Value and Its Treatment Through Life Insurance." This was followed by a presentation by Drs. John Reichel, Peter Masucci, John Boyer and C. H. Barr, of Glenolden, entitled, "A Method of Rapidly Lyophilizing Biological Products."

The following day, papers were presented in the morning by Dr. R. M. Staley, practitioner, of Narbeth, Pa., "A Day's Work

in Small-Animal Practice"; Dr. Frank Breed, of Lincoln, Neb., "Hog Cholera and Allied Infections," and Dr. Frank E. Lentz, "State and Federal Antinaroctic Drug Laws, Rules and Regulations and Their Application to Veterinary Medicine." The final session in the afternoon received reports from Dr. J. G. Hardenbergh, of Plainsboro, N. J., "Veterinary Qualification for Public Health Service"; Hon. J. Hansell French, Secretary of Agriculture, "Progress in the Federal-State Eradication Plan for Handling Bovine Tuberculosis and Bang's Disease," and Dr. W. J. Lentz, "So-called Black Tongue and Allied Conditions."

A. HENRY CRAIGE, JR.
*A. V. M. A. Resident Secretary.*

## CORNELL CONFERENCE FOR VETERINARIANS

A very successful conference for veterinarians was held at the New York State Veterinary College at Cornell University, January 6-7, 1938. This was the 30th annual conference. The attendance was over 300. The program on the first day consisted of a general session in the morning and two sectional meetings in the afternoon. This was followed by alumni meetings in the early evening and an evening program. The morning of the second day was divided into a two-hour period of clinical demonstrations followed by a two-hour, general session. In the afternoon a general session completed the technical portion of the program. The conference was closed by a banquet held in Willard Straight Memorial Hall. The attendance taxed the capacity of the banquet-room. The outstanding event at the banquet was the presentation to Cornell University of the Alumni portrait of Dr. Dennie Hammond Udall. The portrait was accepted by Dr. Edmund E. Day, president of Cornell University.

The papers presented at the conference were as follows:

"Brucellosis in Horses," Dr. W. S. Stone, New York State Veterinary College.

"Filtrable Viruses," Dr. R. E. Shope, Rockefeller Institute, Princeton, N. J.

"The Indigestions of Ruminants, with Special Reference to Treatment," Dr. R. A. McIntosh, Ontario Veterinary College, Guelph, Ont.

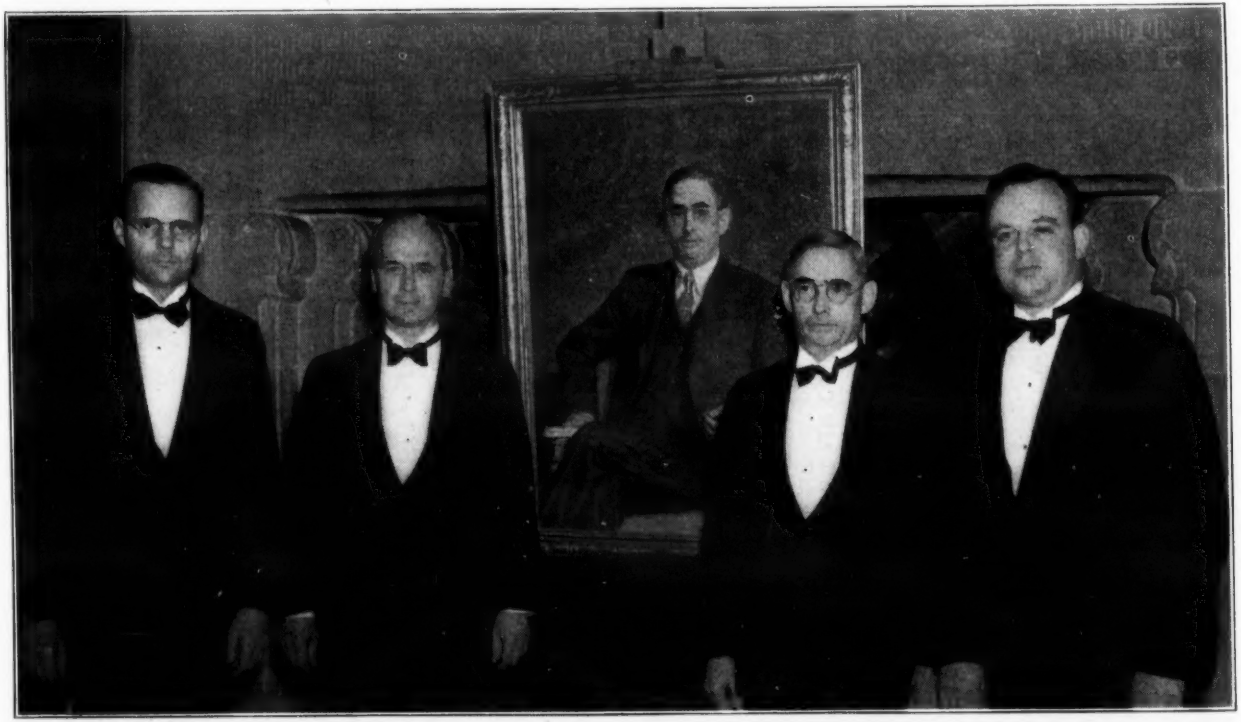
"Traumatic Surgery," Drs. J. N. Frost and A. G. Danks, New York State Veterinary College.

"Some Problems of the Veterinarian in General Practice," Dr. W. J. Gibbons, New York State Veterinary College.

"Swine Influenza and Its Relationship to the Disease in Man," Dr. R. E. Shope.

"Hog Cholera," Dr. Frank Breed, Lincoln, Neb.

"Diagnosis and Control of Parasitic Gastro-enteritis of Calves," Dr. D. W. Baker, New York State Veterinary College.



PRESENTATION OF THE ALUMNI PORTRAIT OF DR. DENNIE HAMMOND UDALL TO CORNELL UNIVERSITY

*(Left to right):* Dr. M. G. Fincher, president of the New York State Veterinary College Alumni Association; Dr. Edmund E. Day, president of Cornell University; Dr. Dennie H. Udall, Professor of Veterinary Medicine, New York State Veterinary College; Dr. William A. Hagan, dean of the New York State Veterinary College.

"Breeding Problems in Dairy Herds," Dr. R. A. McIntosh.

"Producers' Views of Inspection," Dr. A. M. Mills, Plainsboro, N. J.

"Application of the Equine Pregnancy Test," Dr. J. R. Varley, New Brunswick, N. J.

"Changes in the Blood of the Cow at Parturition," Dr. C. E. Hayden, New York State Veterinary College.

"The Production and the Use of X-Rays," Prof. F. K. Richtmyer, Cornell University.

"Canine Dentistry," Dr. C. N. Bramer, Evanston, Ill.

"The Reproductive Cycle in the Bitch and Some Associated Breeding Problems," Dr. H. T. Batt, New York State Veterinary College.

"Nutritional Deficiencies in Growing Swine," Dr. Frank Breed.

"A Discussion of Food and Contact Allergies, with Special Reference to Their Experimental and Natural Occurrence in Lower Animals," Dr. A. F. Coca, Pearl River, N. Y.

"The Rôle of Allergy in Small-Animal Diseases," Dr. H. J. Milks, New York State Veterinary College.

"General Discussion of Allergic Conditions of Animals, with Special Reference to Skin Manifestations," Drs. C. P. Zepp, New York, N. Y., L. A. Corwin, Jamaica, N. Y., and L. J. Desson, Rochester, N. Y.

E. L. BRUNETT, *Reporter.*

## VERMONT VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Vermont Veterinary Medical Association was held at the Montpelier Tavern, Montpelier, on January 8, 1938.

Papers were read by Dr. P. A. Wakefield, on "Acetonemia"; Dr. David Walker, on "Rumenotomy for Foreign Bodies," and Dr. A. A. Mortimer, on "Tetanus in Canines." All of these created good discussions.

The ladies were entertained by Mrs. Ranney and Mrs. Ellis, at the home of Dr. and Mrs. C. C. Ellis. At 6 o'clock, the veterinarians and their wives had dinner together. Mr. LeRoy A. Smith, of Barre, entertained all in a very pleasing manner with readings and character songs.

The Association endorsed the action of the American Veterinary Medical Association in its efforts to secure recognition of the veterinarian in public health work, under Title VI of the Social Security Act.

The election of officers for the ensuing year resulted as follows: President, Dr. David Hopkins, of Brattleboro; first vice-president, Dr. E. K. Treat, of Manchester; second vice-president, Dr. C. M. Miller, of Orwell, and secretary-treasurer, Dr. G. N. Welch, of Northfield.

G. N. WELCH, *Secretary.*

# TENNESSEE VETERINARY MEDICAL ASSOCIATION

The thirtieth annual meeting of the Tennessee Veterinary Medical Association was held at the Hotel Patton, Chattanooga, January 10-11, 1938.

The meeting was called to order by President G. P. Hatchett, of Chattanooga. The invocation was given by Dr. J. L. Topmiller, of Bowling Green, Ky. The address of welcome was delivered by Mr. Ken Whitaker, pinch-hitting for Mayor Ed Bass, of Chattanooga. Dr. Topmiller gave the response.

Dr. Hatchett's presidential address was short, but to the point. He made many suggestions that, if put into operation, would be of great assistance to the veterinary profession and to the Association.

The first paper on the program was presented by Dr. E. L. Shuford, of Asheville, N. C., who discussed "Tonsilitis of the Dog." This paper was of great interest to the small-animal practitioner. The second paper was presented by Dr. W. L. Stroup, of Corinth, Miss., whose subject was "Bot-Campaign Control as Conducted by General Practitioners." This paper created quite a discussion and brought out many ideas for conducting a campaign of this kind.

The election of officers for 1938 resulted as follows: President, Dr. J. I. Siress, of Nashville; first vice-president, Dr. S. H. Woods, of Murfreesboro; second vice-president, Dr. D. Coughlin, of Knoxville, and secretary-treasurer, Dr. A. C. Topmiller, of Murfreesboro. Dr. John H. Gillmann was elected delegate to the A. V. M. A. House of Representatives for a two-year term, with Dr. F. R. Youree, of Lebanon, as alternate.

Col. S. G. Kielsmeier and Dr. J. L. Topmiller were elected to honorary membership in the Association.

The 1938 meeting of the Southern States Veterinary Medical Association will be held in Knoxville, and all members of the Tennessee Veterinary Medical Association are going to assist in every way possible to make this meeting a great success. A motion prevailed that the Association invite the American Veterinary Medical Association to meet in Memphis in 1940. This motion was passed with the understanding that it be agreeable to the veterinarians located in Memphis and the adjoining states.

A clinic for both large and small animals was held at the hospital at Fort Oglethorpe, Ga., with Col. Kielsmeier in charge. This was considered to be one of the best and most constructive clinics that the Association has held in a good many years.

The banquet was held the evening of the first day with Dr. M. Jacob, of Knoxville, acting as toastmaster.

On the second day, Dr. M. Jacob reported on the recent meeting of the United States Live Stock Sanitary Association held in Chicago. Dr. Benjamin Schwartz, Chief, Zoölogical Division, U. S. Bureau of Animal Industry, gave a talk on parasites of interest to the practitioners in the South. Dr. J. M. Sutton, State Veterinarian of Georgia, discussed "Eradication of Bang's Disease by the Area Plan." This paper was of much interest to the regulatory officials and brought out a good discussion. Dr. J. L. Topmiller presented a paper on "Sulfanilamide, Its Usage and Contraindications." This paper proved to be quite instructive.

A. C. TOPMILLER, *Secretary.*

## OKLAHOMA VETERINARY MEDICAL ASSOCIATION

The twenty-third annual meeting of the Oklahoma Veterinary Medical Association was held at the Skirvin Hotel, Oklahoma City, January 10-11, 1938, with a registered attendance of over 100.

At the business meeting held on the morning of the first day, the following officers were elected: President, Dr. H. B. Morgan, of Muskogee; vice-president, Dr. G. A. Cunningham, of Lawton, and secretary-treasurer, Dr. F. Y. S. Moore, of McAlester.

Dr. C. H. McElroy, of Stillwater, was elected delegate to the A. V. M. A. House of Representatives, with Dr. F. Y. S. Moore as alternate.

An amendment to the constitution was adopted which provided for combining the offices of secretary and treasurer in one office. A motion was passed expressing appreciation for the fine publicity program being carried on by the Associated Serum Producers, Inc.

At the afternoon session, a paper was presented by Dr. H. D. Bergman, president-elect of the American Veterinary Medical Association, entitled "Some Newer Developments in Physiology of Therapeutic Significance." Dr. B. M. Lyon, of Pearl River, N. Y., spoke on "Equine Encephalomyelitis" and the discussion of this subject was opened by Dr. H. C. Smith, of Fort Dodge, Iowa. A motion-picture, entitled "The Growth and Development of Bacteria Under Artificial Cultivation," was shown by Dr. Lyon.

At the morning session the second day, Dr. S. W. Haigler, of Saint Louis, Mo., spoke on "Skin Diseases of Dogs," and Dr. Ashe Lockhart, of Kansas City, Mo., presented the subject, "Calfhood Vaccination Against Bang's Disease."

At the afternoon session, the new Oklahoma Veterinary Practice Act, which became effective May 22, 1937, was analyzed and explained by Hon. F. E. Chappell, of Oklahoma City. A paper on "Puppy Problems" was presented by Dr. S. W. Haigler, and Dr. C. C. Hisel, of Oklahoma City, gave reports on the 1937 meetings of the American Veterinary Medical Association and the United State Live Stock Sanitary Association.

A banquet was held the evening of the first day, with an attendance of about 150. Dr. C. H. McElroy acted as toastmaster and Dr. H. D. Bergman was the principal speaker.

The Ladies' Auxiliary provided a program of entertainment for the visiting ladies.

F. Y. S. MOORE, *Secretary-Treasurer.*

## SOUTH CAROLINA ASSOCIATION OF VETERINARIANS

The South Carolina Association of Veterinarians held its annual business meeting, Tuesday evening, January 18, 1938, at the Jefferson Hotel, Columbia, with President H. L. Frieze, of Gaffney, presiding. There were about 55 in attendance, making the meeting one of the largest ever held.

The minutes of the previous meeting were read and adopted. The Secretary was instructed to correspond with the veterinary associations of Georgia, North Carolina and Virginia, relative to suitable dates for holding our summer meeting in Charleston, S. C., during either June or July, 1938.

Dr. W. A. Barnette, of Greenwood, and Dr. B. C. Talley, of Bennettsville, were elected as delegate and alternate, respectively, to represent South Carolina in the A. V. M. A. House of Representatives for a period of two years. Dr. M. R. Blackstock was reëlected to succeed himself as a member of the State Board of Veterinary Examiners. Officers for 1938 were elected as follows: President, Dr. J. H. Moore, of Charleston; vice-president, Dr. D. O. Kitchen (reëlected), of Greenville, and secretary-treasurer, Dr. R. A. Mays (reëlected), of Columbia.

R. A. MAYS, *Secretary-Treasurer.*

## COLORADO VETERINARY MEDICAL ASSOCIATION

The thirty-fourth annual meeting of the Colorado Veterinary Medical Association was held at the Albany Hotel, Denver, January 20, 1938. Dr. T. H. Brady, of La Junta, presided.

After the transaction of routine business, Dr. I. E. Newsom, of Fort Collins, told what he knew of the efforts of the chiropractors to repeal the recently enacted Basic Science Law. He recommended that a resolution be adopted condemning the efforts of chiropractors to interfere with the medical laws of Colorado.

Under the head of new business, the question of inviting the American Veterinary Medical Association to meet in Denver in 1939 was taken up. Dr. Newsom explained why it probably would be inadvisable for the A. V. M. A. to meet in San Francisco in 1939. A motion was carried unanimously, instructing the secretary to prepare a formal invitation inviting the A. V. M. A. to meet in Denver in 1939.

Mr. Frank G. Arnold, a tax expert, gave a very interesting talk on "Taxation in Colorado."

The election of officers resulted as follows: President, Dr. C. C. Stewart, of Colorado Springs; vice-president, Dr. H. L. Franklin, of Greeley, and secretary-treasurer, Dr. J. H. Bouton (reëlected), of Aurora. Members of the Board: Dr. A. N. Carroll, of Pueblo (2 years), and Dr. E. E. Tobin, of Greeley (1 year). Dr. A. N. Carroll was elected delegate to the A. V. M. A. House of Representatives, and Dr. A. A. Hermann, of Denver, as alternate.

Dr. A. H. Francis, of Denver, then made a few pointed remarks regarding the payment, by the federal government, of indemnities on cattle condemned for tuberculosis, stating that an accredited veterinarian should receive authorization to make these tests before doing so. Dr. R. M. Gow, State Veterinarian, also made a few remarks regarding the enforcement of laws requiring veterinary inspection at public sales rings.

Dr. W. A. Sullivan, of Cheyenne, Wyoming, member of the Executive Board of the A. V. M. A. for District 7, made an urgent plea for all eligible veterinarians to become members of the national organization.

J. H. BOUTON, *Secretary-Treasurer.*

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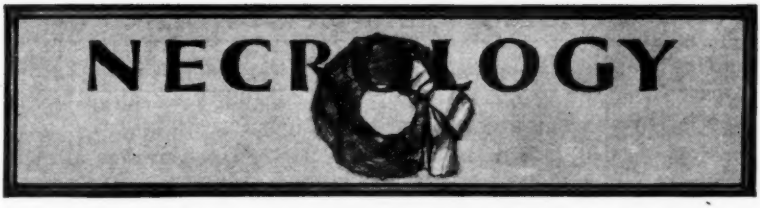
One thorn of experience is worth a whole wilderness of warning.—*Lowell.*




## AARON SILKMAN

Dr. Aaron Silkman died at his home in Goldenbridge, N. Y., August 13, 1937, after a lingering illness caused by a malignant abdominal condition.

Born in Cross River, N. Y., in 1874, Dr. Silkman spent his boyhood on a farm. He studied veterinary medicine at the American Veterinary College in New York City and was graduated with the class of 1895. He engaged in general practice in New York City with his father, who was also a veterinarian, and in 1904 entered the service of the New York City Health Department.

It was due to the work of Dr. Silkman that glanders in New York City was brought under control and practically eliminated by the system of strict quarantine measures, mallein testing, and tagging of horses, which he inaugurated. He was the first one to recognize European fowl plague when it appeared in this country about ten years ago, and it was through his prompt action in establishing quarantine and inspection methods, in coöperation with the federal authorities that this disease was promptly controlled and eradicated. Dr. Silkman retired as chief veterinarian of the Health Department in 1926, and then engaged in poultry inspection work in New York City and Westchester County, N. Y., under the Bureau of Agricultural Economics.

Dr. Silkman was held in high esteem, not only by his associates in the veterinary profession, but by members of the medical profession as well. He was frequently called upon by officials of the Health Department to diagnose transmissible diseases of animals in human subjects. During the later years of his life, he made an intensive study of poultry diseases and it is regrettable that he did not record more of his findings. He was modest and retiring by nature and disliked publicity. He was a life member of Eastern Star Lodge No. 227, Empire Chapter No. 170, R. A. M., and Adelphic Council No. 7. He is survived by his widow and a daughter by a former marriage.

R. S. M.

## FRANCIS MARION COWDEN

Dr. Francis M. Cowden, of Craig, Neb., died on June 2, 1937. Death was the result of complications following an attack of septic sore throat about a week previous.

Born in Greenfield, Ind., July 9, 1882, Dr. Cowden attended high and normal schools before entering the Kansas City Veterinary College. Following his graduation in 1917, he practiced in Oklahoma for a year, spent two or three years in government work, and then located at Craig, Neb., for general practice. Dr. Cowden always was active in civic affairs in his community. He had served on the city council, and at the time of his death was president of the Board of Education. He was a Mason and past master of his lodge.

Dr. Cowden joined the A. V. M. A. in 1924. He leaves to mourn his loss, his widow (née Edith L. Marsolf), two sons, two daughters, his mother and a sister.

E. L. C.

## FRANK P. LAMBORN

Dr. Frank P. Lamborn, of Olathe, Kan., died in the Providence Hospital, Kansas City, Kan., on December 4, 1937, at the age of 58 years. He was a graduate of the Western Veterinary College, class of 1903, and had been in active practice at Olathe until shortly before his fatal illness. He is survived by his widow and one son.

## JOHN PETER BYRON

Dr. John P. Byron died at his home at Britton, S. Dak., on December 8, 1937. He had been in poor health for several years.

Born at Stockholm, Wis., December 2, 1875, Dr. Byron moved to Bristol, S. Dak., with his parents, in 1886. He attended public schools, the Metropolitan Business College in Minneapolis, and the Western Veterinary College in Kansas City. He was graduated in 1906 and practiced in Day County, S. Dak., for 32 years. He had been in the employ of either the state or the federal government on tuberculosis eradication work at various times since 1925. He moved to Britton about two months before his death.

Dr. Byron was elected representative to the State Legislature in 1917, 1933, 1935 and 1937. He held various offices in the City

Council of Bristol and was a member of the Board of Education at the time of his death.

Dr. Byron joined the A. V. M. A. in 1936. He was a member of the South Dakota Veterinary Medical Association. He is survived by his widow (née Nellie Liegenhagen), four sons, four daughters, his mother, four sisters and two brothers.

E. E. F.

## JARL F. OLSON

Dr. Jarl F. Olson, of Cloquet, Minn., died at his home on December 22, 1937, after an illness of about one year.

Born in Finland, on September 9, 1890, Dr. Olson was graduated from the Royal Veterinary High School, at Dresden, Germany, in 1914. He practiced his profession in Finland for about a year, after which time he came to the United States. He was located for a while in Butte, Mont., and moved to Cloquet, Minn., in 1920.

Dr. Olson was a member of the Minnesota State Veterinary Medical Society, and of the Eagle Lodge of Cloquet. He is survived by his widow (née Helmia Backlund), two sons and one daughter.

J .P. F.

## EDWARD B. SIMONDS

Dr. Edward B. Simonds, of Riverdale, Md., died at Daytona Beach, Fla., on December 22, 1937. He had been in failing health for over a year and had been on leave of absence from his duties as U. S. Bureau of Animal Industry Inspector-in-Charge of Maryland and Delaware.

Born at Fayetteville, Ark., June 30, 1878, Dr. Simonds served in the U. S. Army and saw action in the Spanish-American War. He studied veterinary medicine at the United States College of Veterinary Surgeons, in Washington, D. C., and was graduated in 1918. He entered the service of the Bureau of Animal Industry the same year and was assigned to meat inspection. He was transferred to tuberculosis eradication work in Maryland, served as a supervisor for some years, and was made Inspector-in-Charge in 1925.

Dr. Simonds joined the A. V. M. A. in 1928. For the past three years he had represented Maryland in the A. V. M. A. House of Representatives. He was a member of the Maryland State Veter-

inary Medical Association, the United States Live Stock Sanitary Association, the Twelfth International Veterinary Congress and the National Association of B. A. I. Veterinarians. He is survived by his widow (née Delia E. Lester) and three children. He was given a military burial at Arlington Cemetery on December 27.

M. F. W.

## HENRY J. MONGEAU

Dr. Henry J. Mongeau, of Chicago, Ill., died suddenly, December 28, 1937, of a heart attack, while in his hospital at 6448 North Clark Street. He was 55 years old, a graduate of the Chicago Veterinary College, class of 1903, and had practiced in Chicago for 35 years. He was president of the American Cat Association. He is survived by his widow and one son.

## JOSEPH HONORE VILLENEUVE

Dr. Joseph H. Villeneuve, of Montreal, Quebec, Canada, died on December 26, 1937. His varied activities, both professional and civic, had made him one of the most prominent veterinarians in the province of Quebec.

Born in Montreal, January 4, 1886, Dr. Villeneuve received a classical education at Montreal and Saint Mary's Colleges. After a brief but varied career as a sailor, secretary, and superintendent of an animal insurance company, Dr. Villeneuve decided to study veterinary medicine and entered the Montreal Veterinary College. He was graduated in 1915 with the highest average ever attained by a student in the institution. Later, he was a member of the teaching staff of his alma mater.

In 1921, Dr. Villeneuve was appointed city veterinarian of Montreal. For two years he edited *All Dogs*. He founded the Montreal Veterinary College Old Boys Association in 1922, and was president of the Veterinary Medical Association of Montreal from 1921 to 1924. He was director of athletics at the University of Montreal, 1923-1924, and Quebec vice-president of the Amateur Athletic Union of Canada in 1925. Since 1930, he had been official veterinarian to the Quebec Racing Association. He was a life member of the Montreal Kennel Association and for a time was a governor of the College of Veterinary Surgeons of the Province of Quebec.

Dr. Villeneuve joined the A. V. M. A. in 1922 and served as resident secretary for Quebec, 1922-1923. He will be remembered by those who attended the Montreal meeting, in 1923, as the secretary of the Committee on Local Arrangements for that meeting. He was created a Knight of the Order of Agricultural Merit by France in recognition of his services to agriculture. He was a member of the Knights of Columbus and at one time was a warden of Notre Dame Church and secretary of the Notre Dame branch of the Saint Vincent de Paul Society. He is survived by three sons, five daughters, his mother and two brothers.

## PETER A. DILLAHUNT

Dr. Peter A. Dillahunt, of Springfield, Ohio, died on January 1, 1938, in the Springfield City Hospital, where he had been a patient since November 23.

Born in Springfield, Ohio, in 1869, Dr. Dillahunt was graduated from the Ontario Veterinary College in 1893. Except for about three years spent in government service, he had practiced at Springfield until about three years ago.

Dr. Dillahunt joined the A. V. M. A. in 1915. He was a member of the Ohio State Veterinary Medical Association. He is survived by two sons, two daughters, a sister and three brothers. His wife (née Elva Kobelanz) preceded him in death the latter part of October.

## WALTER T. JOHNSON

Dr. Walter T. Johnson, of Corvallis, Ore., died at his home, December 29, 1937, after an illness of almost a year. The cause of death was cancer of the intestines.

Born at Tower, Minn., March 2, 1892, Dr. Johnson moved at an early age to western Washington. After attending local schools, he entered the State College of Washington for the study of veterinary medicine. He was graduated in 1917, and served as instructor in veterinary surgery at Pullman for two years. He then accepted the position of veterinarian to the Western Washington Experiment Station, at Puyallup, where he remained until 1925. He then went to Oregon State College as poultry pathologist on the staff of the Department of Veterinary Medicine.

Dr. Johnson was recognized as one of the outstanding authorities on poultry diseases in this country. He probably was best

known for his contributions to our knowledge of immunity in coccidiosis of poultry. His death interrupts the work of investigating poultry diseases at Oregon State College for which a special appropriation was made at a recent session of the legislature.

Dr. Johnson joined the A. V. M. A. in 1924. He was a member of the 12th International Veterinary Congress and was a reporter on the subject of "Fowl Pox." He was a member of the Washington State Veterinary Medical Association, the American Association of Instructors and Investigators in Poultry Husbandry, Sigma Xi, Phi Kappa Phi and Alpha Psi fraternities. Surviving Dr. Johnson are his widow (née Ethel M. Johnson) his mother, one sister and two brothers.

## GEORGE W. BUTLER

Dr. George W. Butler, of Indianapolis, Ind., died at his home on January 3, 1938. He had been in failing health for several years.

Born in Stirling, Ontario, Canada, March 9, 1858, Dr. Butler was graduated from the Ontario Veterinary College in 1884. He engaged in general practice at Circleville, Ohio, for about 15 years and then entered the service of the U. S. Bureau of Animal Industry. He was assigned to meat inspection in Milwaukee, later being in charge of the stations at Eau Claire, Wis., Louisville, Ky., Lafayette, Ind., and Indianapolis. He retired in 1930.

Dr. Butler joined the A. V. M. A. in 1899. He is survived by his widow (née Ida Kellenberger), one son, a sister and a brother, Dr. Tait Butler (Ont. '85), of Memphis, Tennessee.

C. H. H.

## PAUL T. BURNSIDE

Dr. Paul T. Burnside, of Wellington, Ohio, died while making a professional call, January 4, 1938. A heart attack was given as the cause of death, which came approximately twelve hours after he had been sworn in as a village councilman.

Born on a farm near Mechanicsburg, Ohio, March 1, 1889, Dr. Burnside attended local grade school and was graduated from the Mechanicsburg High School in 1909. He then entered Ohio State University for the study of veterinary medicine. Follow-

ing his graduation in 1912, he practiced at Mechanicsburg for several years. He then entered the service of the State Department of Agriculture, serving in this capacity until 1927. He then returned to Wellington, where he resumed private practice.

Dr. Burnside joined the A. V. M. A. in 1920. He was a member of the Ohio State Veterinary Medical Association. He was prominent in civic and fraternal affairs, being a member of the Wellington Kiwanis Club, the Wellington Grange, an Eagle and a Mason. He was the organizer and adviser of the Wellington 4-H Colt and Calf Club. He is survived by his widow (née Mildred Stillman), one daughter and his father.

## NICHOLAS FERDINAND RATERMAN

Dr. N. F. Raterman, of Fort Loramie, Ohio, died suddenly, January 8, 1938, after an illness of less than an hour. He had just made a professional call at a farm in the community.

Born at Fort Loramie, Ohio, July 15, 1886, Dr. Raterman attended local schools, completed a course at Saint Josephs College, at Rensselaer, and was graduated from the Cincinnati Veterinary College in 1912. He returned to the place of his birth and engaged in general practice right up to the time of his death. For five years he had served as vice-president of the Loramie Banking Company and as vice-president of the Minster Mutual Fire Insurance Company.

Dr. Raterman joined the A. V. M. A. in 1928. He was a member of the Ohio State Veterinary Medical Association and the Northwestern Ohio Veterinary Medical Association. Fraternally, he was identified with the Knights of Saint John. He is survived by his widow (née Henrietta Quigley), a daughter, his mother, three brothers and a sister.

## ARTHUR C. BURRELL

Dr. Arthur C. Burrell, of Indianapolis, Ind., died of pneumonia, in Saint Vincent's Hospital, on January 8, 1938. He had been ill for several months before the fatal attack of pneumonia.

Born in Bloomfield, Ind., in 1888, Dr. Burrell was graduated from the Indiana Veterinary College in 1911. He practiced for several years and then became associated with the Russel M. Seeds Company. About 1917, he established the Burrell-Dugger

Company, manufacturer of poultry remedies, and served as president of the firm since its founding.

Dr. Burrell was a member of the Indianapolis Rotary Club, Indianapolis Athletic Club, Oriental Lodge, F. and A. M.; Murat Temple, Knights Templar, and Elks Lodge. He is survived by his widow, three sons and two daughters.

## EDWARD BONFOY GILLER

Dr. Edward B. Giller, of White Hall, Ill., died suddenly on January 19, 1938, at Our Saviour's Hospital, at Jacksonville, Ill., where he had been a patient for only one day. He had been in poor health and had gone to Jacksonville to consult a physician. The latter had advised him to enter the hospital for observation.

Born at White Hall, July 19, 1886, Dr. Giller had spent practically all of his life in that community. He finished high school in 1905, and then entered the Chicago Veterinary College. He was graduated with the class of 1908 and entered general practice. He later became interested in the breeding of race horses and operated a stock farm near White Hall. He was a director of the Greene County Fair Association, district chairman of the Boy Scouts of America, and had served both as president and director of the White Hall Board of Education. He was a member of the Alpha Psi fraternity.

Surviving Dr. Giller are his widow (née Ruth Davis), two sons, one daughter, his mother, one brother and one sister.

## THOMAS B. HARRIES

Dr. Thomas B. Harries, of Calgary, Alberta, Canada, died on January 4, 1938. He was a graduate of the Ontario Veterinary College, class of 1904, the McKillip Veterinary College, class of 1906, and the University of Pennsylvania, class of 1908. Since his graduation he had engaged in general practice and farming in Alberta. During the World War he served in France with the British Expeditionary Forces, having been attached to the 22nd Divisional Train. Dr. Harries joined the A. V. M. A. in 1909.

Our sympathy goes out to Dr. and Mrs. Truman W. Cole, of Chevy Chase, Md., in the death of their 17-year-old son, Truman W. Cole, Jr., January 23, 1938.

# PERSONALS

## MARRIAGES

Dr. Arthur R. Thiele (K. S. C., '34), of Flushing, N. Y., to Miss Carolyn Gordon Stevenson, of Columbia, S. C., at Columbia, December 9, 1937.

Dr. Edgar W. Holden (Corn. '34) to Miss Eleanor Lois Fields, both of Norristown, Pa., in that city, October 2, 1937.

Dr. Thos. J. Muxlow (K. S. C. '30), of South Saint Paul, Minn., to Miss Mildred Campbell, at Saint Paul, Minn., October 9, 1937.

Dr. Hubert R. Hein (K. S. C. '35), of Greenleaf, Kan., to Miss Marjorie Marie Scott, at Waterville, Kan., October 3, 1937.

Dr. J. D. Jarmon (K. S. C. '21), of New York, N. Y., to Miss Myrtle Price, December 11, 1937.

## BIRTHS

To Dr. and Mrs. Emery H. Enge, of Confrey, Minn., a son, Cyrell Emery, January 17, 1938.

To Dr. and Mrs. A. H. Frank, of College Park, Md., a son, Glynn Henry, September 13, 1937.

## PERSONALS

Dr. C. C. Warkentin (San Fran. '16) has moved from Gilroy, Calif., to Sacramento, same state.

Dr. H. A. Vetterling (Wash. '20) reports a change of address from Wessington to Huron, S. Dak.

Dr. D. H. Bradley (O. S. U. '36) has established a small-animal practice at 4145 North High Street, Columbus, Ohio.

Dr. Stuart M. Dingwall (Wash. '37), formerly of Grants Pass, Ore., gives a new address: 6239 Sycamore Ave., Seattle, Wash.

Dr. G. T. Woodward (Wash. '24), formerly located at Fallon, Nev., is now in Reno, same state. Address: 754 Plumas Street.

Dr. Paul E. Eippert (O. S. U. '36), of Bucyrus, Ohio, has taken over the practice of Dr. J. J. Solt (O. S. U. '36), of Upper Sandusky, Ohio.

Dr. G. H. Lasher (McK. '08), of Rutland, Ohio, was confined to the Holzer Hospital, in Gallipolis, with an attack of tularemia, in December.

Dr. S. W. Stiles (U. P. '31), of Falmouth Foreside, Maine, was assigned to his bed with an attack of undulant fever in the early part of January.

Dr. E. G. Sadler (Corn. '32), formerly in practice at Pine Plains, N. Y., has entered into a partnership with Dr. A. W. Cleaves (McGill '98), at Ellsworth, Maine.

Dr. J. O. R. Campbell (Chi. '07), of Geneva, Ind., is said to have the only veterinary hospital in Indiana equipped with an operating-room for large animals.

DR. DANIEL H. RICKS (A. P. I. '30), of Oklahoma City, has been appointed state veterinarian of Oklahoma, succeeding Dr. C. C. Hisel (K. C. V. C. '16), who resigned.

DR. J. J. SOLT (O. S. U. '36) has sold his practice in Upper Sandusky, Ohio, to Dr. Paul E. Eippert (O. S. U. '36) and returned to Arlington, Ohio, his former home.

DR. H. A. SAILORS (McK. '08), of Wabash, Ind., has been on the sick list for about five months. He has been a sufferer from ascites, incident to cirrhosis of the liver and its complications.

DR. A. F. RANNEY (Corn. '32), of the Bang's disease laboratory, at Montpelier, Vt., was recently assigned to the Augusta, Maine, laboratory for a week to check the work being done there.

DR. G. N. WELCH (Ont. '03), of Northfield, Vt., started on a trip through the sunny South on January 15. He was accompanied by Mrs. Welch and they expected to be gone a few weeks.

DR. JAMES KRAL (K. S. C. '35), formerly located in Albany, N. Y., has entered the service of the U. S. Bureau of Animal Industry and has been assigned to laboratory work at Augusta, Maine.

DR. E. E. SLATTER (O. S. U. '30), who has practiced at Saint Joseph, Ill., for about four years, has purchased the hospital of Dr. J. T. Redmon (Ind. '09), at 1305 East Main Street, Danville, Ill.

DR. MAURICE K. WALTERS (Mich. '34) has been appointed City Meat Inspector of Sturgis, Mich., effective March 1. He succeeds Dr. James L. Boydston (Mich. '37), who has entered practice at Athens, Mich.

DR. JOHN O. F. PRICE (Ont. '91), of Algona, Iowa, was retired from the service of the U. S. Bureau of Animal Industry on November 30, 1937. He had been engaged in hog cholera control in Iowa for a number of years.

DR. W. B. CRAIG (Ont. '89), of Indianapolis, Ind., is in Saint Vincent's Hospital recovering from an operation performed about the middle of January. The operation consisted of the amputation of a leg above the knee.

DR. CHAS. E. FANSLAU (Corn. '17), who was in charge of the Chicago office of the Winthrop Chemical Company, Inc., for a number of years, has been transferred to the main office of the company at New York City, where he is now in charge of the Veterinary Division.

LT. COL. JOHN H. GOULD (Iowa '02), U. S. Army retired, of Des Moines, Iowa, has been in the Army and Navy Hospital for treatment since November 24, 1937. At the most recent report, he expected to leave the hospital soon and be back in Des Moines in March.

DR. J. R. STIFLER (U. S. C. V. S. '11), of Lebanon, Ky., sustained a fractured knee-cap and other minor injuries in an automobile accident on Campbellsville Road, 3½ miles from Lebanon, on January 16. He was taken to a Louisville hospital for treatment.

DR. C. LLOYD TURNER (Ont. '37), who was located at the Animal Diseases Research Institute, Hull, Quebec, Canada, has accepted a three year probationary appointment as Veterinary Officer in the Malayan Veterinary Division of the British Colonial Service, with headquarters at Kuala Lumpur, Selangor, Federated Malay States.

DR. CLIFF D. CARPENTER (Corn. '20) has resigned his position as Director of the Poultry Division of Lederle Laboratories, Inc., to accept a research fellowship at the University of Kentucky. His project is located in the Kentucky Agricultural Experiment Station, at Lexington, where he will devote his time to studies in avian coccidiosis.